TRANSACTIONS

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OTOLARYNGOLOGY

MAY - JUNE
1956

Volume 60 Number 3

ANNUAL MEETING, OCTOBER 14-19, 1956, CHICAGO

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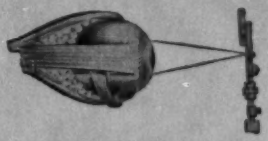

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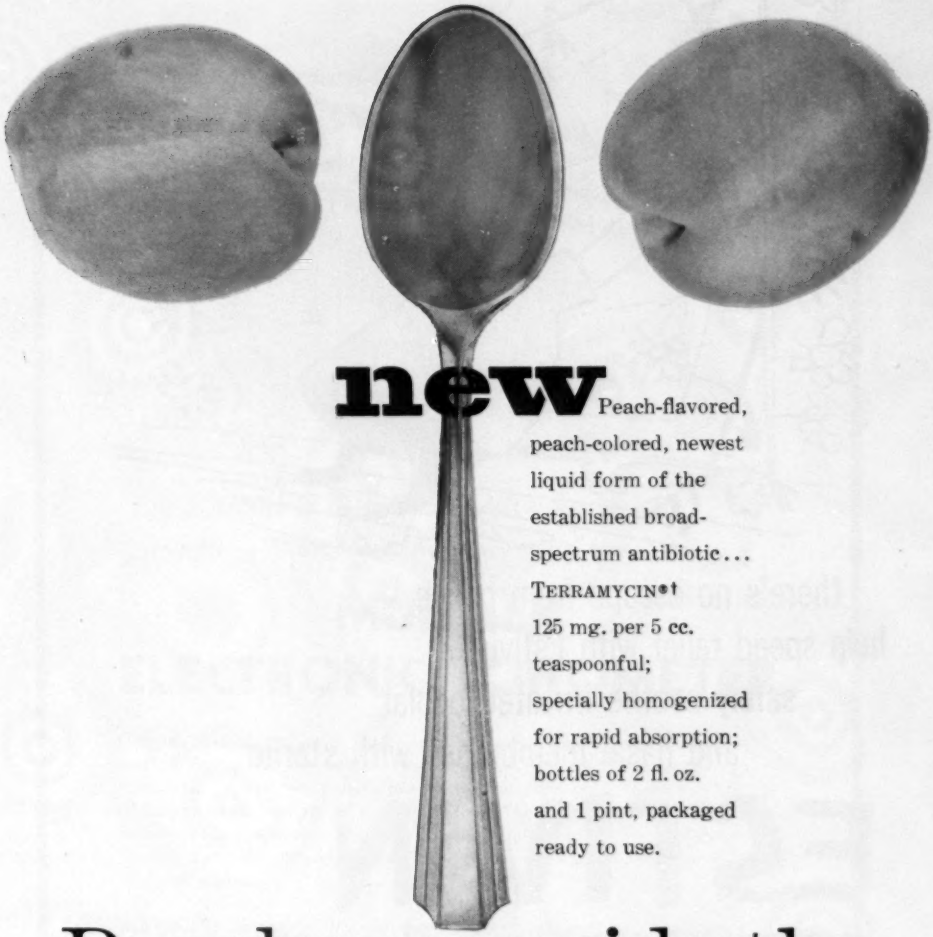

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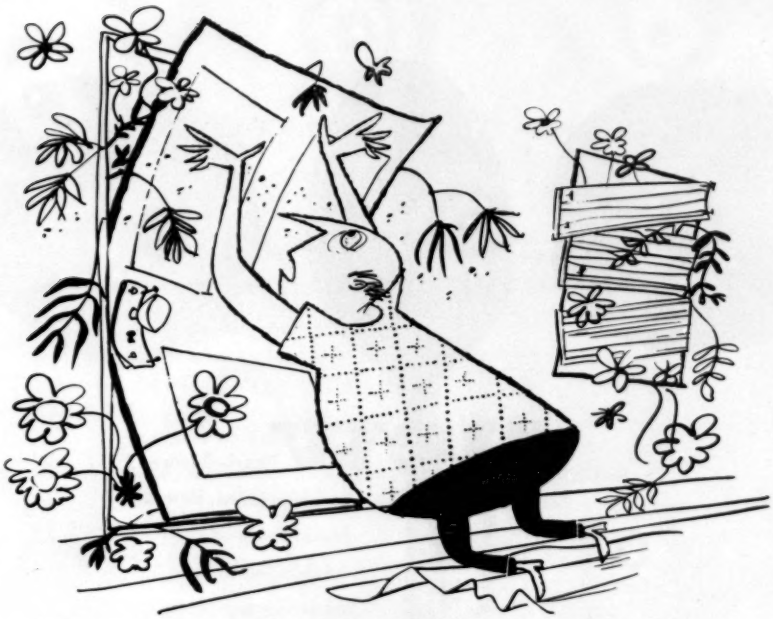

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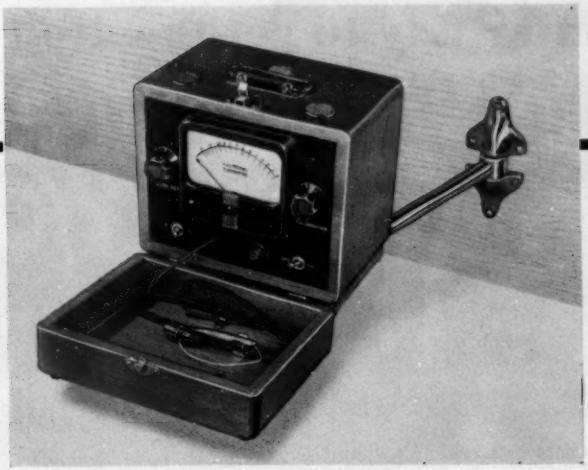

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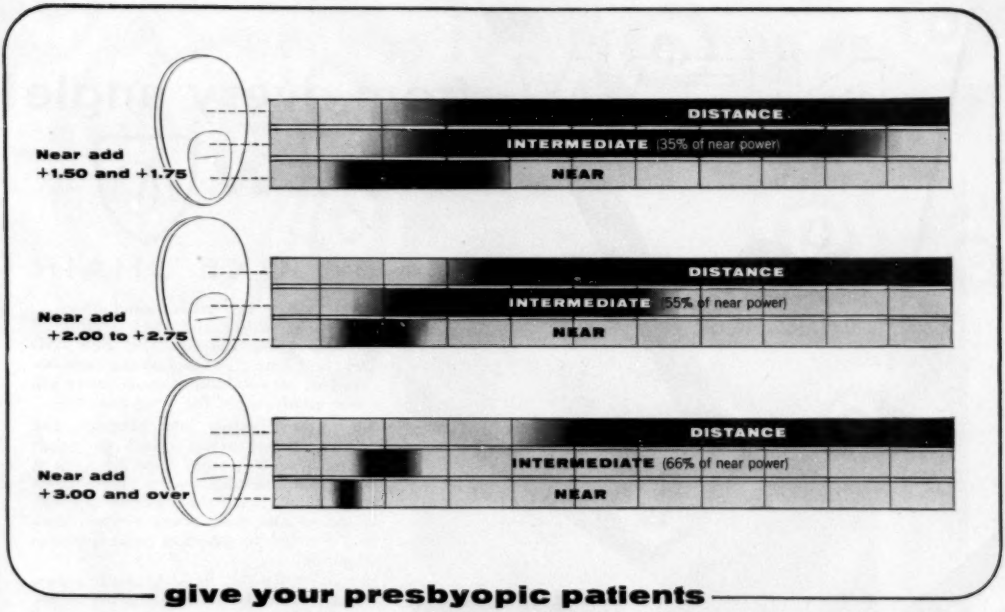

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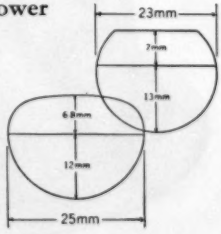

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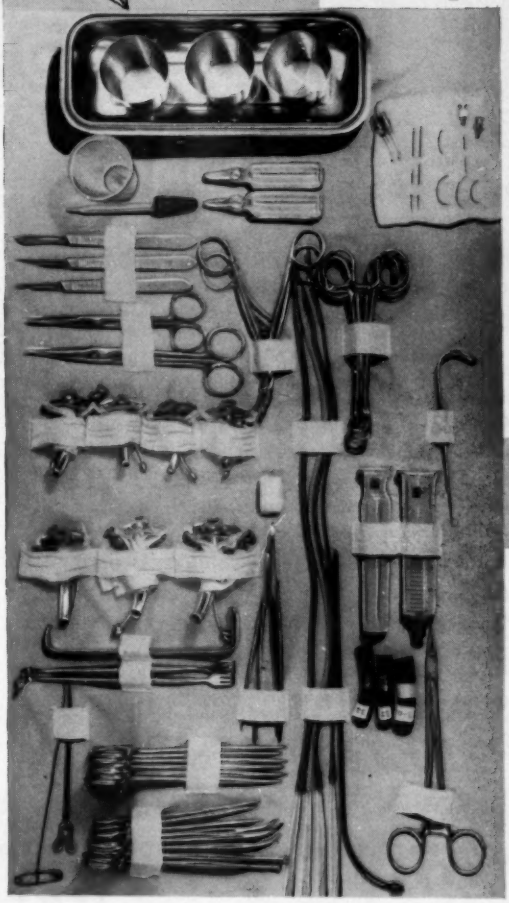

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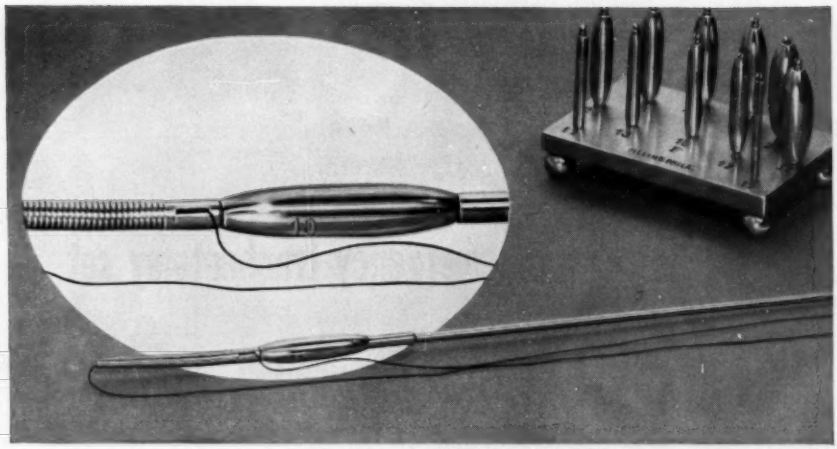

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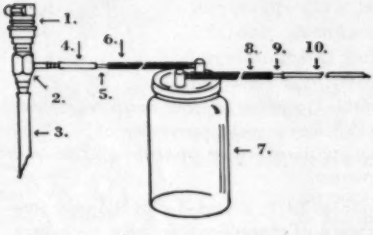

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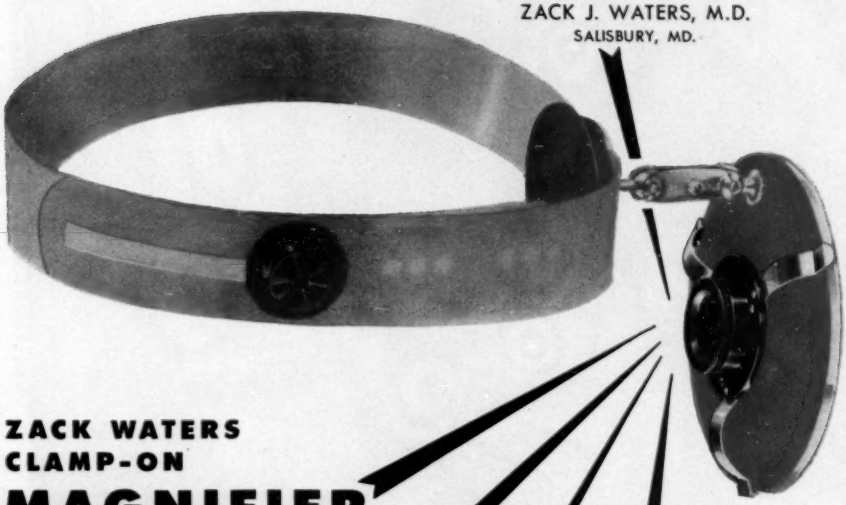

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2. Council on Pharmacy and Chemistry, A.M.A.: New and Nonofficial Remedies, Philadelphia, J. B. Lippincott Company, 1955, p. 263.
3. Gettes, B. C.: A.M A. Arch. Ophth. *51*:467 (April) 1954.
4. Gordon, D. M., and Ehrenberg, M. H.: Am. J. Ophth. *38*:831 (Dec.) 1954 (a review of 8 studies covering 1035 patients).

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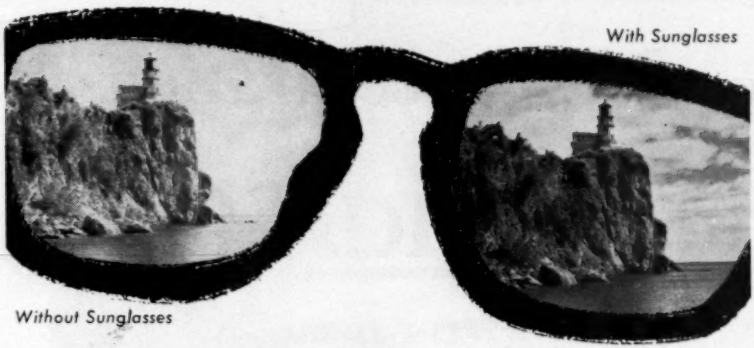

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The Use of the Systemic Hemostat Carbazochrome Salicylate

J. C. Bacala, M.D.

(Abstracted from the Western Journal of Surgery, Obstetrics, and Gynecology, 64:88 (1956)

The author emphasizes the need for dependable chemical control of bleeding and oozing, and reviews the methods that have been tried to attain hemostasis, including the use of vitamin K and bioflavonoids.

He gives the history of carbazochrome salicylate and describes the clinical work completed to demonstrate its effectiveness. It appears to be specific for conditions characterized by increased capillary permeability. It has no effect on large blood vessels; it does not alter blood components; it does not affect blood pressure or cardiac rate. It has neither vasoconstrictor or vasodilator action. The drug has no known contraindications.

He reviews 1051 cases from the files of St. Anthony Hospital, Louisville, Kentucky, of which 330 were treated with Adrenosem Salicylate. 317 were surgical cases and 13 obstetric-gynecological conditions. The treatment was given for symptomatic relief, and as a clinical recourse for patient safety.

In 1015 tonsillectomy patients the following results were noted: There was bleeding in 85 (19.8%) of 429 patients who received no medication. Patients who received a compound of oxalic and malonic acids numbered 115. Of these 16 (14%), developed bleeding. Of 248 patients receiving vitamin K preparations, 47 (19%) had postoperative hemorrhage. 233 patients received Adrenosem Salicylate. Of this group, only 16 (7%) had bleeding.

In other surgical conditions, Adrenosem was used successfully in gastrointestinal bleeding, cataract extraction, incisional seepage, and transurethral prostatectomy. It was also effective in 12 cases of epistaxis.

In the field of obstetrics and gynecology, Adrenosem controlled bleeding in menometrorrhagia, hymeneal laceration, cervical ooze, antepartum and postpartum bleeding, threatened abortion, and in two cases of surgery during pregnancy.

The author points out that in pregnancy, seepage bleeding occurs more because of capillary permeability, and in spite of increased coagulability. Here, Adrenosem becomes specific for strengthening capillary resistance.

Many of the observations in this study, however, involved bleeding incident to trauma of incision, retraction, dissection, tearing and stretching. In these conditions, he reasons that Adrenosem causes hemostasis by its effect on the capillary intercellular cement substance so that the cells draw closer together, and the ends of the capillaries constrict, causing a "self-clamping" mechanism.

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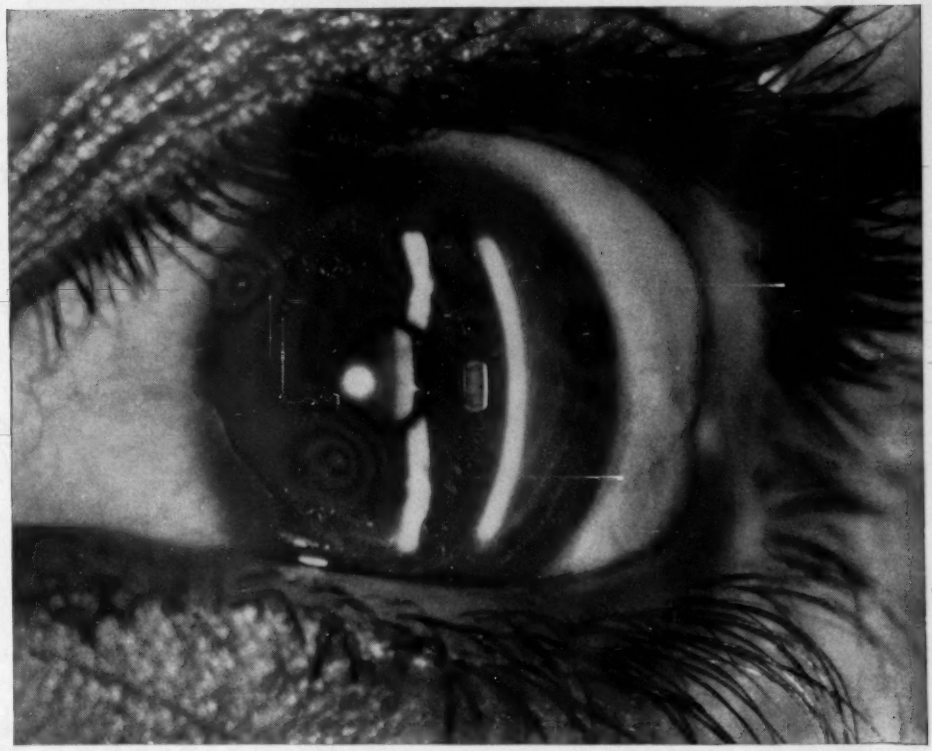

MAJOR ADVANTAGES: Low cost, convenient to use, no systemic effects, local anti-inflammatory effect.

STERILE OPHTHALMIC SUSPENSIONS AND OINTMENTS OF

Cortone® ACETATE (CORTISONE ACETATE U.S.P., MERCK)

HydroCortone® ACETATE (HYDROCORTISONE ACETATE U.S.P., MERCK)

Topically administered hydrocortisone has proved to be "superior [to cortisone] in the following conditions: contact dermatitis of the lids; vernal conjunctivitis; sclerosing keratitis; superficial punctate keratitis; recurrent corneal erosions, and diffuse episcleritis."..."Most other lid and anterior segment diseases responded equally well to either cortisone or hydrocortisone." . . . "The ease of application, relatively low cost, and equal effectiveness on topical use make this method of treatment more desirable."[1]

SUPPLIED: Sterile Ophthalmic Suspensions of CORTONE Acetate—0.5 and 2.5 per cent; 5-cc. dropper bottles. Ophthalmic Ointment of CORTONE Acetate—1.5 per cent; 3.5-Gm. tubes. Sterile Ophthalmic Suspensions of HYDROCORTONE Acetate—0.5 and 2.5 per cent; 5-cc. dropper bottles. Ophthalmic Ointment of HYDROCORTONE Acetate—1.5 per cent; 3.5-Gm. tubes.

Reference: 1. Hogan, M. J., Thygeson, P. and Kimuras, J., *Arch. Ophth.* **53**:165, Feb. 1955.

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DIVISION OF MERCK & CO., INC.

Here he can judge quality for himself

but he depends on your judgment for LENS quality

He's a good judge of quality in the ordinary things he buys and uses for work and play. But when he needs lenses, he relies on your judgment of quality.

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WHITE OR TONETEX®

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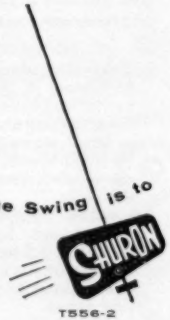

See the SHURON vision care message in the June 9 issue of the **POST.**

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Introducing

ANOTHER DOHO "FIRST"

exciting new

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BACTERICIDAL AND FUNGICIDAL WITHOUT SIDE EFFECTS

SOOTHING THROAT SPRAY/GARGLE

For infectious and non-infectious sore throat involvements as:

- smoker's throat
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- post-nasal drip
- tense, non-salivating throat found in actors, singers and artists

for your "sandpaper throat" patient

Easy-to-use Larylgan Throat Spray supplies medication in a convenient plastic squeeze-atomizer, **at no additional cost to the patient.**

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in ocular surgery

THORAZINE*

"not only prevents vomiting,

but results in cheerful, more relaxed patients and ophthalmic surgeons."[1]

'Thorazine' is available in ampuls, tablets and syrup (as the hydrochloride), and in suppositories (as the base).

*T.M. Reg. U.S. Pat. Off. for chlorpromazine, S.K.F.

in ocular surgery, 'THORAZINE'

produces "a more relaxed patient, and an uneventful post-operative period"

'Thorazine' produces "a calmer, more co-operative patient. . . . barbiturates given alone may make a patient drowsy even to the point of sleeping, but do not diminish the fear of and reaction to the pain . . . nor do they allay pre-operative anxiety. ['Thorazine'] seems to produce a more relaxed patient, and an uneventful post-operative period."[2]

lessens the need for patient co-operation

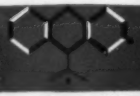
"The main advantages of ['Thorazine' premedication] are: (1) no co-operation is required from the patient during the operation or for several hours afterwards; (2) it produces a low-tension eye ideal for intraocular surgery; and (3) the recovery period is extremely tranquil."[3]

controls nausea, vomiting and anxiety

"There was no nausea, vomiting or anxiety, somnolence was pronounced and amnesia was noted frequently. Complications attributable to 'Thorazine' were completely absent.

"It is safe to conclude that 'Thorazine' . . . is an extremely valuable addition to preoperative medication in patients to be done under local anesthesia."[4]

Smith, Kline & French Laboratories, Philadelphia 1

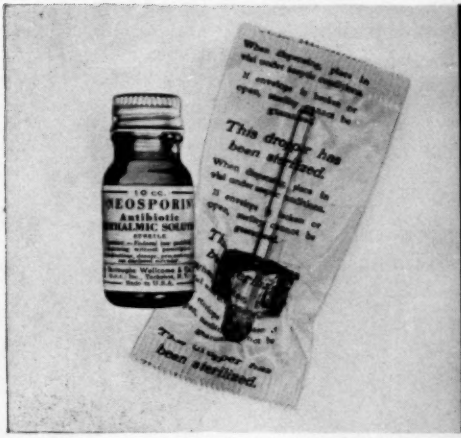


1. Becker, B.: Chlorpromazine—A New Anti-Emetic Agent, Am. J. Ophth. *38*:576 (Oct.) 1954.
2. Moore, J. G.: Chlorpromazine (Largactil) as a Premedication in Ophthalmic Surgery, Brit. J. Ophth. *39*:109 (Feb.) 1955.
3. Nutt, A. B., and Wilson, H. W. J.: Chlorpromazine Hydrochloride in Intraocular Surgery, Brit. M. J. *1*:1457 (June 18) 1955.
4. Fritz, M. H.: Thorazine® as Preoperative Medication in Ophthalmology and Otolaryngology, Eye, Ear, Nose & Throat Monthly *34*:515 (Aug.) 1955.

Announcing a new solution for eye infections

'NEOSPORIN'® brand Antibiotic OPHTHALMIC SOLUTION

guards eyes meticulously against bacteria



sterile

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rapidly effective

Each cc. contains:

'Aerosporin'® Sulfate Polymyxin B Sulfate	5,000 Units
Neomycin Sulfate (Equivalent to 1.75 mg. Neomycin Base)	2.5 mg.
Gramicidin	0.025 mg.

Bottles of 10 cc. with sterile dropper.

Also available—for dermatologic and ophthalmic infections:

'NEOSPORIN'® brand Polymyxin B–Bacitracin–Neomycin ANTIBIOTIC OINTMENT

Tubes of 1 oz., ½ oz. (with applicator tip), and ⅛ oz. (with ophthalmic tip).

Literature available on request.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N. Y.

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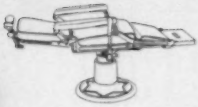
by

Reliance



Shown above is the #650 Reliance Chair, a foot hydraulic model of outstanding quality.

On the right is the #550 Treatment Chair with its Convenient single lever control for raising and lowering, locking the revolving action and reclining the chair (see small pen sketch).

May we send you information about these and the many other fine Examination and Treatment Chairs produced by Reliance?

F. & F. KOENIGKRAMER CO.

Manufacturers since 1898

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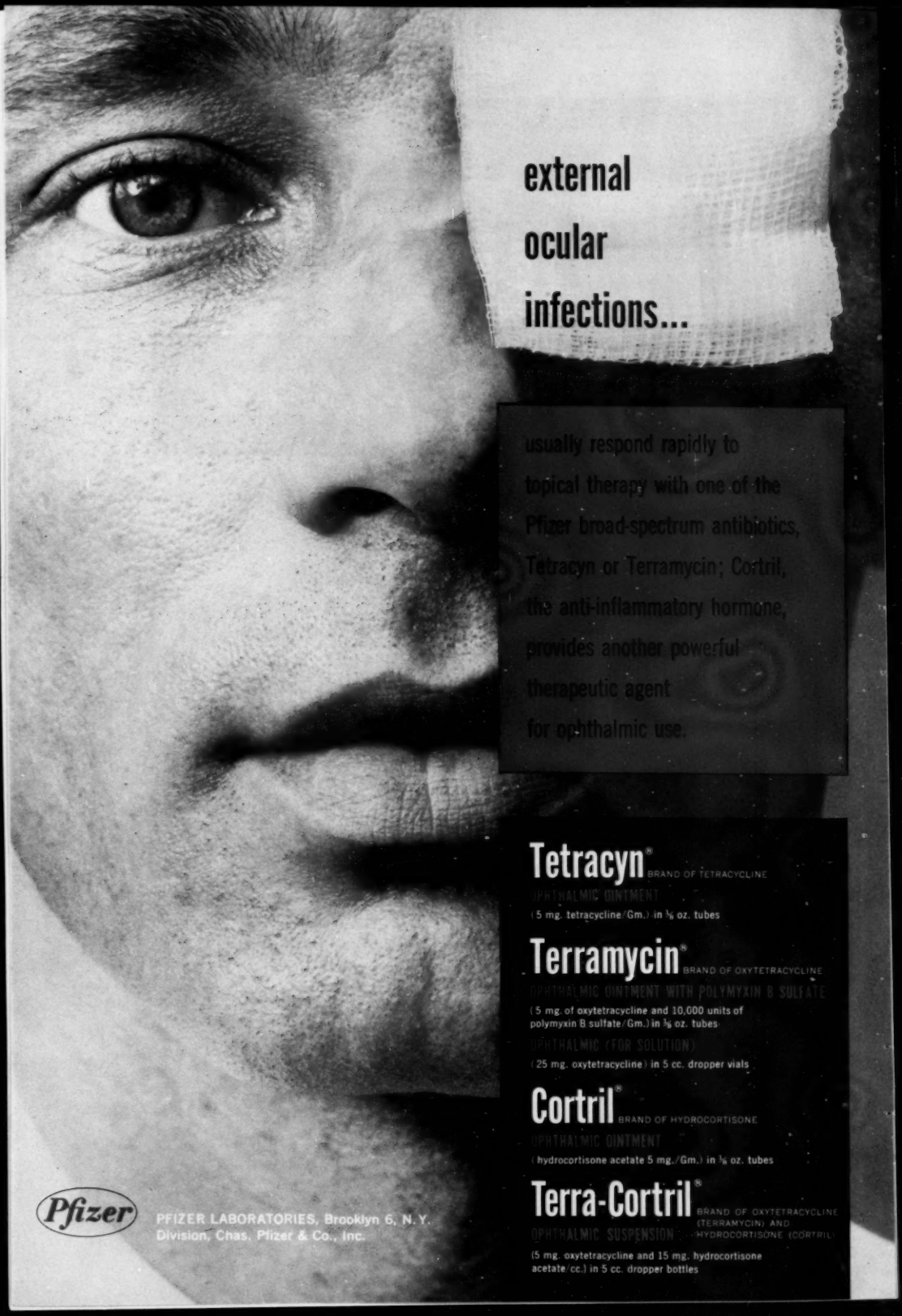
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usually respond rapidly to
topical therapy with one of the
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provides another powerful
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for ophthalmic use.
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OPHTHALMIC (FOR SOLUTION)
(25 mg. oxytetracycline) in 5 cc. dropper vials
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(hydrocortisone acetate 5 mg./Gm.) in ⅛ oz. tubes
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OPHTHALMIC SUSPENSION
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"LACRILENS"® is a safe lens

The test of time proves that the "LACRILENS"® contact lens can be worn for long periods of time in aphakia and in all sports, including swimming.

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Embryology of the Ear

MOTION PICTURE SLIDES

OPHTHALMIC PATHOLOGY

ATLAS of PATHOLOGY

OTOLARYNGIC PATHOLOGY

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Neuro-ophthalmology — Leinfelder	Plastic Surgery — Becker	Hearing Tests — Juers
Glaucoma — Meyer	Description of the Orbit — Fralick	Larynx — Pressman
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STERILE SOLUTION in
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One drop dilates pupil fully in less than 20 minutes.
Pupil returns to normal within 2 hours.
Can be used on patients of all ages.

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Surgical Instruments

For
Use with
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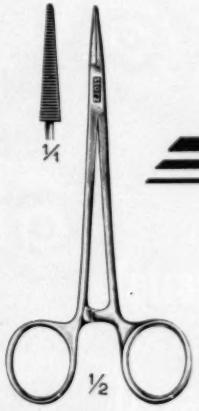

Forceps, *Hemostatic, Mosquito,*
non-magnetic for use with magnet in removing of foreign bodies in ocular surgery, delicate model, with narrow tips.....Each $ 7.50
Six for $40.00

Storz Instrument Company
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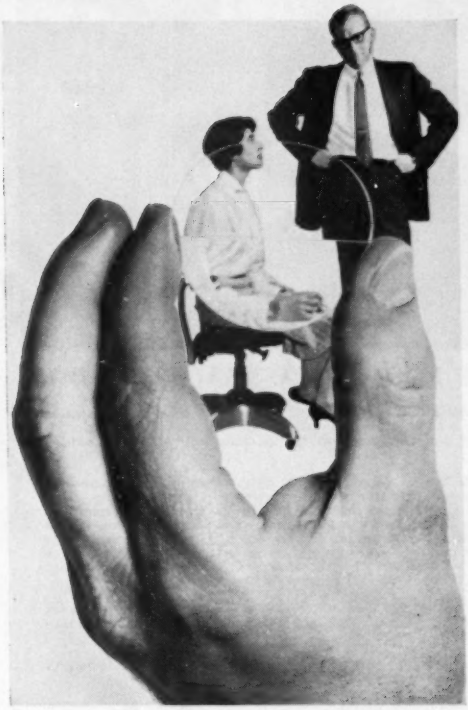
The Last Word in Versatility

TILLYER

EXECUTIVE

BIFOCAL



The fundamental optical design for the Tillyer Executive Bifocal dates back 250 years to Benjamin Franklin. But, to American Optical must go the credit for fully developing this unique lens design and creating an exclusive bifocal lens which has no peers for versatility—The Executive.

The Executive solves many of the problems you face with bifocal patients—especially, first-time wearers. Here are some of the major reasons:

Image jump is completely eliminated.

The wide segment affords unparalleled fitting flexibility. Segment is full width of lens with a maximum height of 28mm.

The Executive incorporates the Tillyer Principle of corrected curves for maximum marginal correction.

The one-piece design reduces color aberrations to a minimum.

If you aren't already prescribing Executives, give them a try—you *and* your patients will be glad that you did.

American Optical

A COMPLETE TREATMENT ROOM IN A SMALL AREA

The dimensions in the above photograph are 5′ 9″ along one wall and 4′ 2″ along the other. In this area, conveniently arranged, is everything needed to make a complete examination. However, it need not be confined to this limited space. The complete absence of piping and wiring is especially noticeable. A number of other arrangements will be furnished on request.

Treatment Stand: Covered with stainless steel. Contains: air regulator with gauge, tubing, cutoff and air filter; nasopharyngoscope rheostat; push button; cautery transformer and rheostat; transilluminator complete with rheostat, cord, handle, lamps, antrum and frontal tips; seven metal drawers on fiber runners and one large drawer for towels; waste container; bakelite covered work table; space for six spray bottles, twelve solution bottles, shelf for cotton jars; pocket for history card or prescription blanks; and a spray bottle heater; the whole presents an attractive appearance and will not tarnish and is easy to keep clean. Price complete without glassware $195.00. Without air but with electrical equipment $165.00. With air equipment only $150.00. Without either air or electrical equipment $115.00. Glassware available.

Chair: Can be placed in horizontal position, will not tip in this position; raised and lowered by large hand wheel; paper covered headrest removable; locked with foot-pedal; bakelite armrests removable. Back locks rigidly in any position. As shown in brown or green. $150.00.

Turning Chair: By adding a footrest and the necessary handles this chair can be used for turning tests. All the additional equipment is easily and quickly removable. Add. $30.00.

Combined Suction and Cuspidor: This is an entirely new piece of equipment. It was designed to eliminate the cost of installing a separate suction. The apparatus is 12″ wide and projects 13¼″ from the wall. The flushing rim and the suction is furnished with a vacuum breaker and should meet all plumbing requirements. Stop and trap not furnished. Available in ivory, green and white. Price $95.00.

Ceiling Light: A telescopic light attached to the ceiling. May also be attached to base board in back of chair. Available in 4, 5, 6 and 7 ft. (extended). 4 ft. $16.50 (add one dollar per ft.).

Light Shield: Takes an ordinary 100 watt lamp. Will not burst; spot can easily be rotated. $5.00.

Stool: A soft upholstered stool with easy running ball bearing stem casters. $20.00. Stool with back, $26.00.

All the equipment shown in this advertisement, ready to be attached to your air system, $531.50.

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All Prices are F.O.B. Los Angeles, Calif.

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inflammatory, allergic eye disorders

prompt relief

in acute episodes

continuing benefits

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METICORTELONE*

(PREDNISOLONE)

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- up to 5 times as potent as hydrocortisone
- minimizes sodium retention, edema, weight gain
- spares patients salt-poor diets

tablets supplied in 3 strengths—1 mg., 2.5 mg., 5 mg.
—for convenient, individualized therapy.

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Eddie Kerr has helped physicians fit thousands of patients with artificial eyes. Like the other experts from our offices, he provides you with technical information and assistance on difficult cases. The samples in his case will match many patients. Or, he can make eyes that perfectly match with the materials he carries. One of our experienced men visits most areas regularly—another reason to call or write our nearest office for your next ocular prosthesis.

Complete Glass and Plastic Artificial Eye Service

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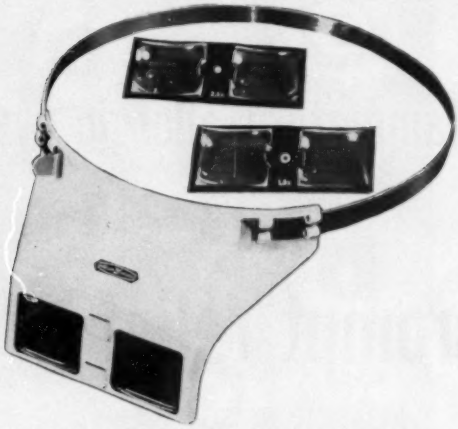

Permits interchange of higher or lower power lens mountings. Designed to give complete comfort when wearing glasses. Can be sterilized without harm. Now available with choice of 1½X, 2X or 2½X. **$12.50** P.P. Additional powers **$5** pr. ppd.

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TERRA-CORTRIL®

brand of oxytetracycline and hydrocortisone

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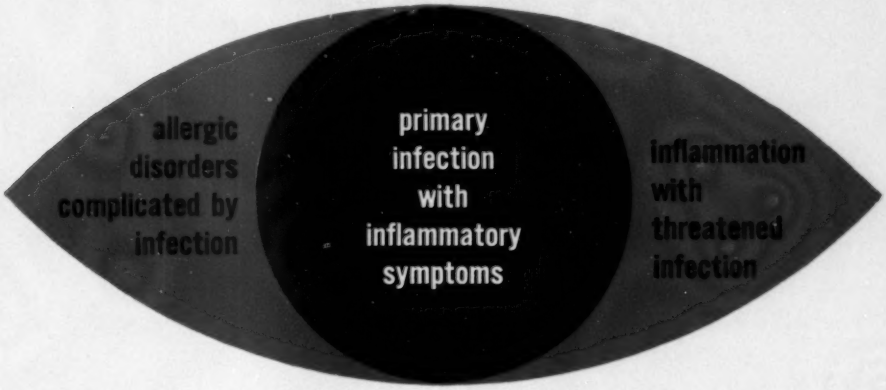
allergic disorders complicated by infection
primary infection with inflammatory symptoms
inflammation with threatened infection

provides the combined action of CORTRIL,® potent adrenocortical hormone, and TERRAMYCIN,® highly successful broad-spectrum antibiotic, for a variety of organisms encountered in ophthalmologic practice.

SUPPLIED: in amber bottles of 5 cc., with sterile eye dropper, containing 5 mg. oxytetracycline hydrochloride (TERRAMYCIN) and 15 mg. hydrocortisone acetate (CORTRIL) per cc. of sterile suspension. Also available: CORTRIL Acetate Ophthalmic Ointment and CORTRIL Tablets.

PFIZER LABORATORIES Pfizer Division, Chas. Pfizer & Co., Inc. Brooklyn 6, New York

for otitis

'AEROSPORIN'® SULFATE

POLYMYXIN B SULFATE WITH PROPYLENE GLYCOL

OTIC SOLUTION STERILE

Specifically aimed at aural pathogens—

bactericidal to most gram-positive and gram-negative organisms, particularly *Ps. aeruginosa*, the commonest cause of otitis externa.

fungicidal to most of the dermatomyces found in the ear.

For otitis externa, whether acute or chronic, an exceptionally high percentage of complete clearance in a short time.

For chronic otitis media (when the ear drum is perforated); preferably in conjunction with systemic therapy.

Bottles of 10 cc. (with dropper)

Burroughs Wellcome & Co. (U.S.A.) Inc., Tuckahoe 7, New York

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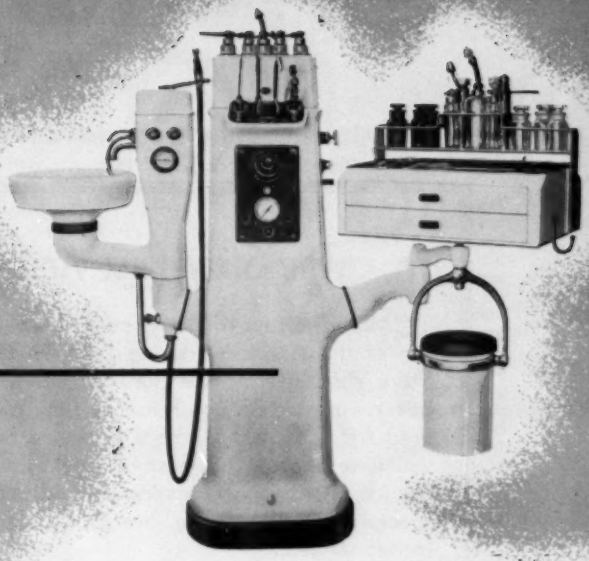
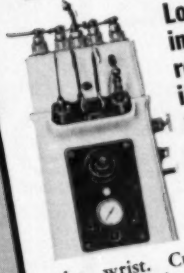


Medicament, special spray and suction bottles within easy reach

Swinging instrument table easily positioned where you want it with a touch of the fingers. Stainless steel tray holds sterilized instruments, plus any additional electrical instruments in readiness.



Low voltage instruments ready for instant use

Low voltage instruments positioned for easy grasp, are used without constant cord pull, yet are returned to the unit with a flick of the wrist. Current automatically turned off when instrument returned to holder.

Individual rheostats control cauteries and illuminated instruments. Auxiliary switch can control room lighting or Ritter Compressor. All controls and call button are within easy reach . . . pressure and vacuum gauge visible from operating position. Unit has extra plug-jack on side, for attaching head lamp or other low voltage instruments.

You have *everything* at your fingertips! . . . with a Ritter Ear, Nose and Throat Unit. With this unit, equipment necessities for ENT work are carefully placed within your examining and treatment area . . . the cuspidor, conveniently located for patient use . . . the surgical aspirator with interchangeable tips right at hand . . . the low voltage instruments and spray bottles, centrally located for easy grasp . . . a swinging tray, holding medicaments and other instruments, effortlessly positioned to suit your needs.

The Ritter Ear, Nose and Throat unit was designed to help you utilize your skills to the fullest extent with a minimum of effort. You have your choice of four models—the cuspidor on the right or left side as an integral part of the unit, or a right or left-hand unit with the surgical cuspidor separate for placing on the opposite side of the chair.

WRITE for complete, detailed information on the Ritter ENT Unit, Ritter ENT Motor Chair, or Ritter Combination Ear, Nose and Throat-Oral Surgery Unit. Ritter Company Inc., 4818 Ritter Park, Rochester 3, New York.

Ritter Company Inc.

The Sixty-First Annual Convention

More than six thousand persons will register for the annual meeting of the Academy during the five days, Sunday through Friday. More than twenty-five hundred Fellows, more than one thousand guests, more than one hundred fifty residents, more than one hundred fifty candidates, more than one hundred orthoptic technicians will take part in the most comprehensive educational program ever conducted for ophthalmologists and otolaryngologists. Some of the special events will be oversubscribed and early registration is recommended.

THE TEACHERS' SECTION: Sunday, October 14, 10:30 a.m., in the Grand Ballroom. A combined meeting of the Ophthalmologic and Otolaryngologic Teachers' Sections. President Furstenberg; Ruedemann, Hoople and eminent guest speakers will discuss the future standing of Ophthalmology and Otolaryngology in Medical Education.

PLASTIC SURGERY: Tuesday, 6:30 p.m. Dinner at the CONGRESS HOTEL, Glass Hat. $7.50 per plate. Order dinner tickets in advance. Your order will be acknowledged and your ticket placed in your advance registration envelope. All orders must be accompanied by check to cover cost of ordered tickets.

ANNUAL DINNER: Palmer House, Grand Ballroom, Monday, October 15, 7:00 p.m. Entertainment—dancing—no speeches—excellent steak dinner. Ten dollars—**seats reserved.**

LADIES ENTERTAINMENT: Look for special announcement.

EXHIBITS: Scientific exhibits on the Club Floor, Foyer and Rooms 15, 16, and 17. Technical exhibits in Exhibition Hall and Red Lacquer Rooms.

INSTRUCTION COURSES: Orders are now being filled.

CLINICS IN MEXICO: A postconvention tour of Mexican cities with visits to the outstanding hospitals of Mexico City—tour capacity limited — regular scheduled flights — no chartered planes — a delightful trip to interesting cities and resorts of old Mexico.

Hotel reservation in Chicago for the week of the meeting should be secured now. Write the Chicago Convention Bureau, 33 North La Salle Street, Chicago 2; give your order of choice of three hotels, type of room desired, date of arrival and departure.

American Academy of Ophthalmology and Otolaryngology

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XII Jackson Memorial Lecture

FAILURE IN CONGENITAL CATARACT SURGERY: A STUDY OF FIFTY-SIX ENUCLEATED EYES

FREDERICK C. CORDES, M.D.
SAN FRANCISCO, CALIFORNIA

IT is indeed an honor to have been asked to give the Edward Jackson Lecture. Dr. Jackson, who was born in 1856 and died in 1942, was one of the really great figures in ophthalmology. In 1888 he became professor of diseases of the eye at the Philadelphia Polyclinic Hospital, and in 1890 he was appointed surgeon at Wills Eye Hospital. Because of the poor health of his wife, he moved to Denver in 1894, and it was this move that introduced him to the Far West, where he was destined to play such an important part in ophthalmology. When Dr. John Weeks also established himself in the West, in Portland, he and Dr. Jackson kept in close touch with each other, and their warm, lively friendship was a source of pleasure to them both as long as they lived.

Dr. Jackson's accomplishments are too well known to need reiteration, but I should like to mention two of his activities. He took a prominent part in the organization of the American Board of Ophthalmology and was its first president, and in 1917 he became the first editor of the third series of the *American Journal of Ophthalmology*, which combined a number of small publications.

Dr. Jackson joined the Pacific Coast Oto-Ophthalmological Society in 1911 and attended every one of its meetings until 1939. In the course of these meetings it was my great privilege to become well acquainted with him. He was a modest man, always liberal in praise of the work of others, always genial and friendly, an indefatigable worker, and a great teacher who was interested above all in the young men in ophthalmology. The warmth of his personality was never more apparent than during the early morning walks so many years ago when it was my good fortune to accompany him through the grounds of the old Del Monte Hotel. His words of advice and encouragement to me as a young man were never-to-be-forgotten gems.

And so I feel that indeed it is a great honor for me to be allowed to present this Edward Jackson Lecture.

From the E. S. Heller Laboratories of the Department of Ophthalmology, University of California Medical Center, San Francisco.

Acknowledgement is made to Dr. Kenneth Swan, Dr. Algernon B. Reese, Dr. Alston Callahan, Dr. Frederick Carriker, and the Armed Forces Institute of Pathology for some of the material and histories used in this paper. Particularly, I wish to express my thanks to General Elbert DeCoursey and Dr. Lorenz Zimmerman of the Armed Forces Institute of Pathology, who made it possible for me to obtain 32 case histories and sections of eyes from the Institute.

Presented at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

INTRODUCTION

It has been known for many years that the surgery of congenital cataracts does not offer as good a prognosis as the surgery of cataracts in the adult. As long ago as 1805, James Ware, in his book Chirurgical Observations Relative to the Eye, made the statement, "Operation is not so certain a cure for cataract

in children as in persons of more advanced age." A certain percentage of the failures can be attributed to congenital anomalies that interfere with the restoration of vision; others are the result of well-recognized early and late postoperative complications. Clinical observation suggests that certain of these complications may be associated with the type of operative procedure employed. In a search for histopathologic confirmation of such an association, a study has been made of 56 eyes removed after surgical failure.

The operations employed for the correction of congenital cataracts are (1) simple discission, usually repeated a number of times; (2) through-and-through discission (Ziegler operation); (3) discission followed by linear extraction; (4) simple linear extraction; and (5) linear extraction with limbal incision and iridectomy, with or without sutures. Optical iridectomy is used so rarely that it is not considered in this discussion.

The Complications of Congenital Cataract Surgery and Their Possible Relation to Type of Surgery

The most frequent complications of congenital cataract surgery are glaucoma, pupillary displacement or occlusion, and retinal detachment. Seen less frequently are corneal changes (e.g., edema and bullous keratitis), epithelization of the anterior chamber, uveitis, sympathetic ophthalmia, atrophy of the globe, phthisis bulbi, endophthalmitis, and panophthalmitis.

Glaucoma. The causes for this complication, which have been reviewed by Chandler,[3] are as follows:

1. A rapid swelling of the lens which pushes the iris forward and blocks the pupil. This is most likely to occur after discissions in which the opening in the capsule has been too small.

2. The retention of a large amount of cortex in the anterior chamber. This can occur in discission or in linear extraction. When it occurs in linear extraction,[4] opening the wound and washing out the cortex controls the glaucoma.

3. Blocking of the pupil as a result of iris bombée. This complication occurs when the pupil is not well dilated, or as a result of considerable inflammatory reaction.

4. The formation of peripheral anterior synechias as a result of delayed restoration of the anterior chamber. This complication occurs in linear extraction and can be prevented if the incision is made at the proper angle so that a valve-like opening is established. The injection of air before the patient leaves the table is also an important preventive. The importance of restoring the chamber at the time of operation has been stressed by Otto Barkan.[2]

Chandler[3] regards incarceration of iris or lens material in the wound as another frequent cause of peripheral anterior synechias.

5. Blocking of the pupil by vitreous and filling of the anterior chamber by vitreous. This complication as a cause of secondary glaucoma has been stressed by Velhagen.[19] It may occur after linear extraction when there has been vitreous loss, but it is most likely to occur after repeated discissions. At times the vitreous completely fills the anterior chamber; in this event it is probably a factor in the production of peripheral anterior synechias. If linear extraction is properly performed, there is little danger of vitreous loss.[5]

Pupillary displacement or occlusion. The principal causes of pupillary dis-

placement, as given by Huerkamp,[11] are prolapse of the iris, contusion of the iris, and prolapse of the vitreous. Huerkamp found that in 52 per cent of cases that had had five operations, there was pupillary displacement. In this series he found that the greater the number of operations the higher the percentage of pupillary displacement.

Occlusion of the pupil may result from the formation of a thick membrane composed of capsule and lens material, or of a complicated membrane due to inflammation, organized exudate, or hemorrhage. Velhagen[19] stated that in evaluating a membrane or aftercataract it was difficult to differentiate between proliferation of the capsule and the formation of an inflammatory pupillary membrane. He advised that these membranes or aftercataracts be operated upon by discission with deWecker scissors so as to prevent pulling on the ciliary body, which could cause hemorrhage in the anterior chamber. Denig,[7] Chandler,[3] and Cordes[4] have all stressed the fact that failure to do an iridectomy in those cases in which the pupil will not dilate preoperatively may be another factor in the production of occlusion of the pupil. Horay[10] felt that the small, bound-down pupil was probably the chief cause of postoperative complications; therefore, he advised that iridectomy be done in every case. In a personal discussion, Chandler stated that when the eye was very small and the pupil would not dilate well preoperatively, he did an iridotomy below in addition to the iridectomy, since the pupil might draw up in such cases if an iridectomy alone were done.

Velhagen[19] pointed out that as long as there is cortical material in the area of the discission, injury to the posterior capsule (vitreous) breaks up resorption and is one of the causes of inflammatory pupillary membrane formation.

Another cause of membrane formation in the pupillary area which may occlude the pupil or produce posterior synechias is the discontinuance of mydriatics before all the cortex is absorbed.

Retinal detachment. This important postoperative complication has received a good deal of attention. As stated by Doggart,[8] an important consideration to be borne in mind is the predisposition of the aphakic eye to retinal detachment. The association of detachment with aphakia is not always apparent, however, since twenty years or more may elapse before the retina detaches and by that time the patient is likely to be under the care of another ophthalmologist. Foster Moore[14] felt that removal of the lens, whether by extraction or discission, predisposed to development of detachment of the retina. Doggart[8] went so far as to state that enough evidence had accumulated to suggest that an eye deprived of its lens had at least one chance in four of sustaining retinal detachment within two or three decades.

Denig,[7] Barkan,[2] Cordes,[4] Doggart,[8] and others have all maintained that retinal detachment is most apt to be a late complication. Shapland[17] found that the average interval between discission and the onset of detachment was 24.6 years. A number of reasons for this delay have been given. Denig[7] feels that delayed resorption of lens material could lead to recurrent irritation of the uvea, which might produce a delayed detachment. Huerkamp[11] found that the pupil was displaced in 54 per cent of the cases of retinal detachment.

Most authorities agree with Chandler[3] that the more the vitreous is disturbed, the greater the danger of retinal detachment as a late complication. Vitreous loss at the time of operation, with vitreous incarcerated in the wound, is apt to result in cicatrization, later in contraction, and eventually in detachment

through traction.[17] Owens and Hughes[16] state that the type of operation relates significantly to postoperative complications, and Chandler,[3] Barkan,[2] Horay,[10] Knapp[13] and others all agree that late detachment occurs much more frequently after needling, especially repeated needling, than after linear extraction. Foster Moore[14] found that 7 per cent of all eyes operated upon for congenital cataract by needling developed retinal detachment; Shapland[17] found the figure to be 10.7 per cent against 2.2 per cent in senile cataract. In 22 patients reporting to Moorfields with retinal detachment after needling, 40 eyes had been operated upon; at the end of an average of 24.6 years, there were 33, or 82.5 per cent, with retinal detachment.

Neame[15] felt that the operation devised by Ziegler should be abolished, since detachment was more likely to follow it than other procedures, and this opinion was concurred in by Spaeth.[18] Cridland[6] agreed that any procedure that caused disturbance of the vitreous was a bad thing. He found glaucoma and detachment in a series of eyes which had undergone Wheeler discissions, and he emphasized that in performing a capsulotomy for an aftercataract or secondary membrane, the minimum procedure should be adopted which would produce a central opening.

In his series Shapland[17] found that progressive myopia must be considered as a possible etiologic factor. Trauma accounted for 13.6 per cent of his cases. As a possible explanation for the evolution of the detachment, he suggested further that in a certain percentage of cases the repeated needlings, especially when applied to a dense capsule, produced traction on the zonule and caused a retinal dialysis; that the retinal margin flapped inward toward the vitreous, thus hindering reattachment, and that then, years later, as the vitreous slowly underwent degenerative changes, the more fluid portion got behind this flap and initiated a retinal detachment in the usual manner. Holes are found only rarely in these cases (in 15.2 per cent of Shapland's series), and the operative prognosis is usually very poor.

Corneal edema, bullous keratitis, and vascularization of the cornea develop in some of these cases as a result of other severe complications. As a more direct association, Velhagen[19] suggested the possibility that corneal dystrophy might occur if vitreous touched the posterior surface of the cornea after vitreous loss or after a breaking of the hyaloid which would permit vitreous in the anterior chamber. Denig[7] also noted that there seemed to be some association between retinal detachment and corneal epithelial dystrophy.

Phthisis bulbi is another complication of congenital cataract surgery. Huerkamp found it in 69 per cent of the eyes that developed "severe complications" postoperatively. Velhagen[19] felt that the pulling action of the capsule forceps in attempts to remove a shrunken cataract or a very dense aftercataract could cause a good deal of damage to the ciliary body which would result later on in phthisis bulbi. Kiss[12] made the interesting observation that the usually harmless iridectomy for prolapsed iris is a dangerous procedure, predisposing to phthisis bulbi, when performed in the first year of a child's life.

The Relation of Type of Surgery to Clinical Results

Clinical experience with congenital cataract surgery suggests a definite relationship between the type of surgery and the quality of the results achieved. The reports of Falls,[9] Horay,[10] Owens and Hughes,[16] Bagley,[1] Chandler,[3] and others would seem to indicate that some form of linear extraction is the opera-

tion of choice. If the pupil can be dilated easily preoperatively, the simple linear extraction would seem to be preferable; if the pupil is difficult to dilate, iridectomy followed by linear extraction is indicated.

Experience has shown that no one operation can be recommended to the exclusion of all others. In cases of membranous cataract, shrunken cataract, or aftercataract, linear extraction quite obviously is not indicated because of the danger of vitreous loss when an attempt is made to grasp the capsule. There is added danger in the fact that in such cases there are often adhesions to the vitreous face. These cases can readily be diagnosed on the basis of a deep chamber and iridodonesis. Some form of discission, with the least possible damage to the vitreous, is then indicated.

A STUDY OF 56 EYES REMOVED AFTER CONGENITAL CATARACT SURGERY

In an attempt to determine, on a histopathologic basis, the relationship between failure in congenital cataract surgery and the type of operation employed, 56 eyes were studied microscopically. Forty (71.6 per cent) of the 56 had been enucleated after needling, 14 (25 per cent) after linear extraction and 2 (3.4 per cent) after operations the type of which had not been recorded (table I). Of the eyes in which a needling had been done, 29 (72 per cent) had had multiple needlings; 11 (25 per cent) had had a single needling, one of which had had a Ziegler through-and-through operation (table II). One of the linear extractions followed a needling.

Table I

Fifty-Six Eyes Enucleated After Failure of Congenital Cataract Surgery

TYPE OF SURGERY	NUMBER OF CASES	PERCENTAGE
Discission	40	71.6
Linear extraction	14	25
Type of surgery unknown	2	3.4

Table II

Forty Eyes Operated Upon by Discission

TYPE OF DISCISSION	NUMBER OF CASES	PERCENTAGE
Multiple discission	29	72
Single discission	10	26
Ziegler through-and-through	1	2

Table III

Glaucoma Present in 26 Eyes (46 Per Cent of All Cases)

TYPE OF SURGERY	NUMBER OF CASES	PERCENTAGE
Multiple discission	16	61.5
Single discission	5	19.4
Ziegler through-and-through	1	3.8
Linear extraction	4	15.3

POSTOPERATIVE COMPLICATIONS

Glaucoma

Of the 56 eyes removed, 26 (46 per cent) had had glaucoma. Of this group, 21 eyes had been removed after needling, 4 after linear extraction, and one after the Ziegler through-and-through operation (table III). Of the 21 glaucomatous eyes enucleated after needling (52 per cent of the eyes needled), 16 (71 per cent) had had from two to six needlings. The 4 glaucomatous eyes that had had linear extraction represented a total of 27 per cent of all linear extractions. Thus, in this series, glaucoma was very much more frequent after needling, especially after multiple needlings, than after linear extraction.

Factors leading to the development of glaucoma. There was occlusion of the angle in 20 of the 26 glaucomatous eyes, and all but 2 of them were eyes enucleated after needlings. In most instances there were several contributing and usually related factors underlying the glaucoma.

1. *Uveitis* was a contributing factor in many cases, as evidenced by the presence of anterior synechias that obliterated the chamber angle. There was one case (no. 5) of absolute glaucoma. In some instances there were also posterior synechias, e.g., case 35, and in still others a considerable part of the iris was adherent to the posterior corneal surface, e.g., cases 7, 8, and 27.

2. *Buphthalmos* was present in 5 cases, all of which had been subjected to multiple needlings. The patient in case 7 had a normal eye until after a second needling at 12 months, when glaucoma and buphthalmos developed. In case 11, that of a 25-year-old man, the buphthalmos developed after several "operations" in childhood, and the eye was enucleated because of the pain of absolute glaucoma. Case 27 was that of a 12-year-old boy whose eye was normal in size until after the second needling when he was 4 years of age, when it began to enlarge. In case 30, that of a 9-year-old boy, there was marked enlargement of the anterior segment with an intercalary staphyloma; the mother was not sure after which of several needling operations the changes had started. Case 43 was that of a 7-year-old boy with rubella cataracts. The left eye started to increase in size when the child was 2 years of age, after the second needling. The right eye, which also had a congenital cataract, had a single needling, five months after the birth of the child, without complications.

3. *Intraocular hemorrhage* was the cause of glaucoma in 10 eyes. Eight of these had had multiple needlings. In 2 cases the hemorrhage resulted from trauma and can therefore be excluded from the present discussion. Two cases followed surgery for glaucoma that developed after cataract surgery: in case 4 an iridencleisis had been attempted in connection with a linear extraction; in the other case the operative hemorrhage followed a third discission.

4. *Spontaneous hemorrhage* occurred in 5 cases. In cases 5 and 6, massive subchoroidal hemorrhages developed 20 years after multiple needlings. In case 43, massive recurrent hemorrhage occurred 50 years after multiple needlings; the eye had had an old detachment of the retina. In case 21, in which there was recurrent hemorrhage four years after surgery, a mass of proliferated epithelial tissue, containing blood and needle-like spaces (cholesterol slits), could be seen in the angle inferiorly; superiorly, the angle was open but narrow.

5. *Pupillary membrane* of a dense nature occurred in 6 cases. In case 4, in which a linear extraction had been followed by an unsuccessful iris inclusion operation, there was a fibrous membrane extending inward from the anterior chamber angle meshwork and the iris. In case 17, that of a 14-year-old boy, a severe uveitis developed after a discission and the eye was enucleated for fear of sympathetic ophthalmia. Examination revealed an inflammatory pupillary membrane adherent to the anterior surface of the iris in addition to the degenerated lens substance. There was a massive anterior capsular cataract. In the 4 other cases, there was membrane formation with traction on the ciliary body and retina, which will be discussed later. Two cases warrant special comment.

6. *Block of the pupil by vitreous.* In case 21 the eye developed glaucoma after the second discission. The pupil was completely occluded, with vitreous pro-

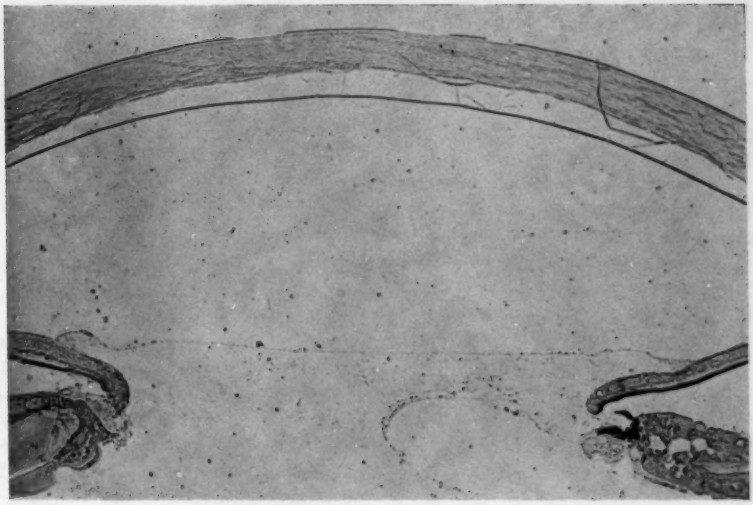

FIG. 1—Pupil occluded with vitreous prolapse and adhesions to iris surface.

lapse and subsequent adhesions of vitreous to the iris (fig. 1). Gonioscopic examination revealed a completely blocked angle. In microscopic section the vitreous appeared in the form of a thin pupillary membrane adherent to the anterior iris surface (fig. 2). In addition, there were posterior synechias to the lens remnants behind the iris in the form of a Soemmering ring.

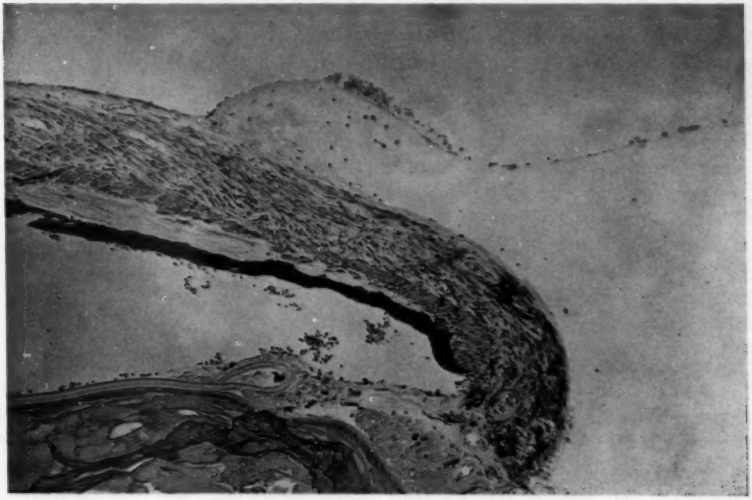

FIG. 2—High magnification of anterior iris surface to show vitreous adhesions.

In case 42, that of an 8-year-old boy, the second discission was followed by hemorrhage and glaucoma. Microscopic examination revealed iris bombée with peripheral anterior synechia. A cuticular membrane was seen on the anterior border layer at the site of the operative wound; it extended to the pupillary margin of the iris, to become continuous with a delicate membrane which stretched across the pupillary opening to the opposite iris leaf, to which it was adherent. The iris was adherent to the lens remnants. The cuticular membrane, probably a condensed vitreous layer, extended backwards through the central opening to the lens capsule.

It was Chandler[3] who particularly directed our attention to these cases. The development of glaucoma following congenital cataract surgery may easily be overlooked because it is not suspected and because of the difficulty of examining the eyes of children with congenital glaucoma under the slit lamp. Although it can develop after linear extraction when there has been vitreous loss, it occurs most frequently after repeated discissions. If a small pupil is completely blocked by a membrane, the vitreous, when cut into, may come forward and again block the pupil. As the pressure increases in the posterior chamber, an iris bombée may be produced, the vitreous being forced into the anterior chamber, sometimes entirely filling it. As Chandler[3] points out, the possibility of this complication is another reason why von Graefe's dictum, "Never do a needling in the presence of a small pupil," should be observed.

7. *Glaucoma after Ziegler through-and-through discission.* Case 9 is a most instructive one. This patient, a 22-year-old woman, was subjected to a Ziegler through-and-through discission. Three days later the eye became red, painful and hard; the diagnosis was that this was an anaphylactic reaction. An attempt to remove the cortex through a keratome incision was unsuccessful and accompanied by vitreous loss. Following surgery, the eye developed endophthalmitis and detachment of the retina, with loss of light perception. The resultant soft, degenerated, sightless eye was enucleated.

Microscopically, the eye did not present the picture of phacoanaphylaxis described by Verhoeff and Lemoine, so that in all probability the glaucoma was caused by the rapid swelling of the lens and resultant blocking of the angle. Since it is almost impossible to avoid vitreous in the anterior chamber after a Ziegler operation, the removal of lens substance without vitreous loss and its attendant complications, if glaucoma should develop, becomes difficult, if not impossible. This case bears out the clinical observation that the possibility of this complication is one of the real disadvantages of the Ziegler operation.

8. *Epithelial downgrowth.* A downgrowth of epithelium may be a factor in the production of glaucoma. Case 35 was that of a 29-year-old man who had had multiple needlings. He developed a red, painful eye that did not respond to therapy and was enucleated. Examination of the sections showed the iris incarcerated in a surgical scar on one side. There was a downgrowth of epithelium which had encircled the posterior chamber and had adhered to the posterior iris surface and to the anterior surface of the Soemmering ring (fig. 3). One mass of epithelial cells was anterior to the iris (fig. 4).

Detachment of the Retina

Detachment of the retina occurred in 23 of the 56 cases (41 per cent); 18 of these followed needling; 4 followed linear extraction; and one occurred 10 years after congenital cataract surgery

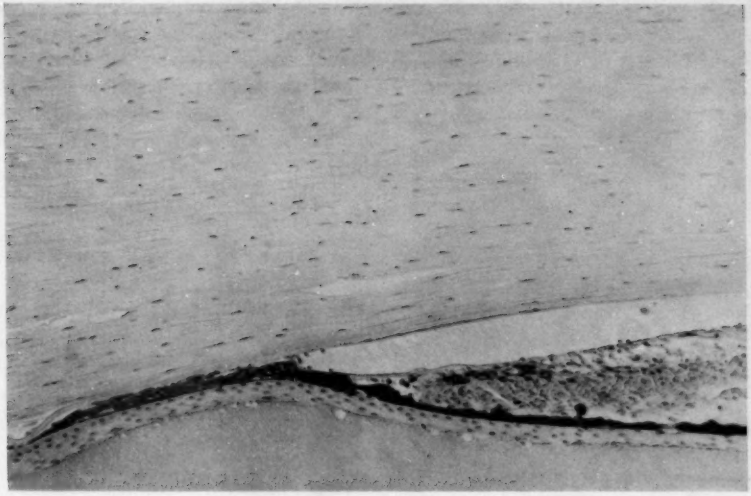

FIG. 3—Downgrowth of epithelium in anterior chamber.

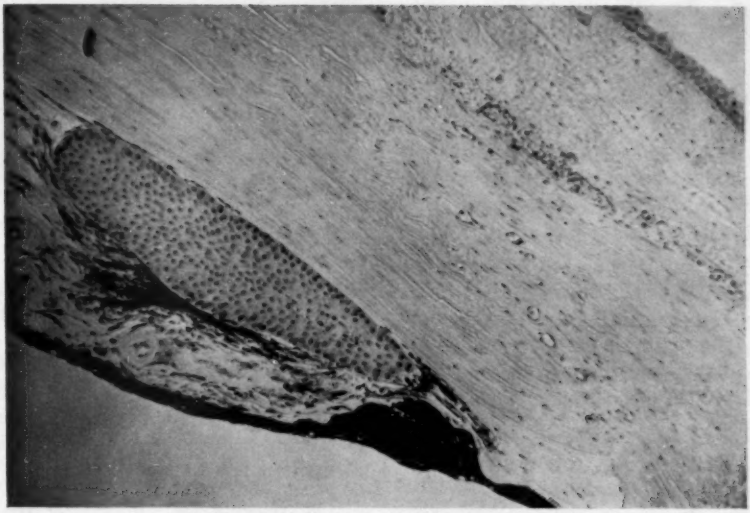

FIG. 4—Epithelial cells between iris and cornea.

of unknown type (table IV). In one patient the detachment developed immediately after a single needling, and in another case it developed soon after a linear extraction. In another patient it followed the cutting of an aftercataract with deWecker scissors.

TABLE IV

DETACHMENT OF RETINA IN 23 EYES (41 PER CENT OF ALL CASES)

TYPE OF SURGERY	NUMBER OF CASES	PERCENTAGE
Discission		
Single	5	21.7
Multiple	13	56.5
		78.2
Linear extraction	4	17.3
Unknown	1	4.5

In one patient (case 3) a discission was done for congenital cataract, and postoperatively there was a white mass in the pupillary area which was diagnosed as retinoblastoma. The eye was enucleated, and microscopic examination showed the mass to be an old retinal detachment.

There were 13 cases of detachment that followed multiple needlings; this was 45 per cent of all the cases that had multiple needlings. In case 24 the condition developed immediately after the third needling.

In case 48, the patient was struck in the eye after the second of two discissions. This resulted in a detachment of the retina associated with recurrent intraocular hemorrhage. The eye developed a chronic uveitis and became atrophic.

Of particular interest are the 12 cases that developed late detachment, 11 after multiple discissions and one twenty-six years after a linear extraction (table V). The longest interval between operation and detachment was in case 43, in which the detachment developed thirty-three years after the last needling (fig. 5). In some instances it was difficult to determine the exact time of the detachment, but so far as could be determined, the average interval after surgery for this group of 11 cases was 22.2 years. This corresponds closely with Shapland's clinical finding of an average of 24.6 years between needling and detachment.[17]

TABLE V

LATE DETACHMENT OF THE RETINA (12 CASES)

TYPE OF SURGERY	NUMBER	PERCENTAGE
After multiple discission	11	37.9 per cent of all multiple discissions
After linear extraction	1	7.1 per cent of all linear extractions
Total	12	

Average time between surgery and detachments, 22.2 years.

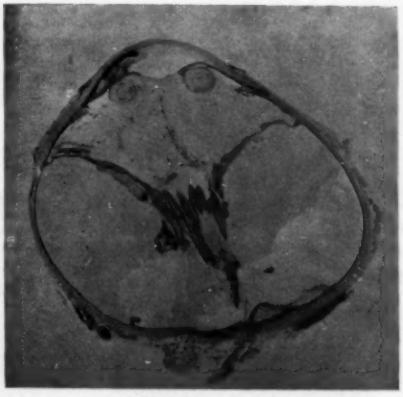

FIG. 5—Retinal detachment occurring thirty-three years after last needling.

The reasons for enucleating the 23 eyes with detachment are interesting. In

7 cases there was extensive recurrent intraocular hemorrhage followed by phthisis bulbi. In three instances phthisis bulbi occurred without hemorrhage following chronic inflammation. In one there was a severe uveitis that developed after an unsuccessful attempt at retinal detachment surgery; in another there was a marked secondary glaucoma that did not respond to therapy.

One 24-year-old woman (case 5), who had had multiple discissions when she was 4 years of age, developed a retinal detachment and an endophthalmitis that went on to absolute glaucoma, which necessitated enucleation.

Of the 3 linear extractions with detachment of the retina, the eye was enucleated in one instance because of the degenerative changes, including bullous keratitis, that followed the cutting of an aftercataract by deWecker scissors; in the second case the cause of enucleation was absolute glaucoma with organized hemorrhage accompanied by an intercalary staphyloma. Absolute glaucoma was also the indication for enucleation in the third case.

In the one case in which the type of surgery was unknown, the detachment was followed by phthisis bulbi and a painful eye.

Shapland's theory, as given above, that traction may sometimes be a factor in the production of detachment, seems to be substantiated by several of the cases in this series that followed multiple needlings (fig. 6). In case 14 there was a cyclitic membrane near the pupillary area which incorporated both the posterior surface of the iris and a portion of the completely detached retina (fig. 7). In one eye, case 43, after three needlings, some delicate strands of tissue extended across the pupillary space and connected with a membrane on the anterior surface of the iris and with bands

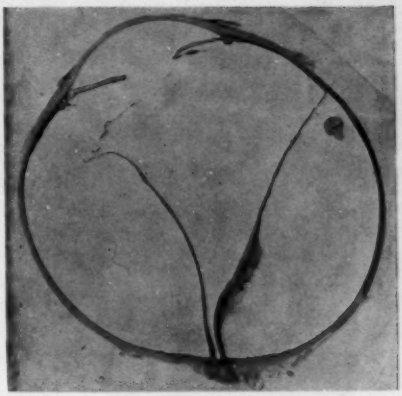

FIG. 6—Late detachment of retina, with strands extending back to detached retina.

of tissue extending to the ora serrata above, below, and backwards to the detached retina, which was ruptured just above the disc. In case 44 there had been five needlings; at a point where an incision had been made to needle the lens there was a downgrowth of fibrous tissue which extended back into the posterior portion of the globe; by contracting, it had pulled on the retina and caused a detachment of both retina and choroid (fig. 8).

In case 37, in which multiple needlings had been followed by an iridectomy for glaucoma, connective tissue extended from the deeper aspects of the scar into the anterior chamber to become continuous with lens, iris, and detached retina. Similar changes were present in case 55, that of a patient who had also had multiple needlings.

These findings would seem to explain, at least in part, why the operative prognosis for retinal detachment surgery is so poor in these cases.

In case 50, a linear extraction had been done thirty-six years before enucleation. Data on the time the detachment had occurred were not available, but from the appearance of the retina

FIG. 7—Late detachment of the retina after multiple needlings (whole eye in figure 5).

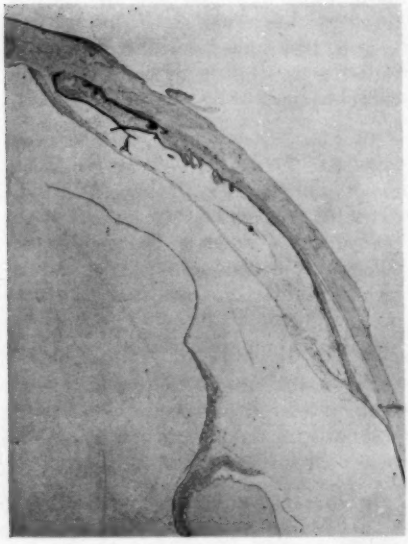

FIG. 8—Portion of downgrowth of fibrous tissue from incision to detached retina.

and the presence of ossification it can be assumed that the detachment had been present for some time prior to enucleation. The blind, phthisical eye was enucleated because of a severe iridocyclitis. Microscopic examination revealed a fibrohyaline cyclitic membrane extending from the operative scar to the iris, lens remnants, ciliary body, and detached retina.

In case 8, enucleated two and a half years after surgery, a detachment followed a linear extraction. The nerve head was pulled forward and gliosed; it contained laminated and apparently calcific drusen. Spontaneous hemorrhage was apparently another reason for enucleation.

In 5 cases of late detachment that occurred fourteen, fifteen, sixteen, twenty, and twenty-two years after multiple needlings, the detachment was apparent-

ly caused by spontaneous subretinal hemorrhage; in case 48, however, the detachment followed extensive intraocular hemorrhage five months after the second needling.

Uveitis

There were 13 cases (20.8 per cent) in the series in which uveitis was at least one of the outstanding symptoms. Seven of these occurred in patients who had had multiple needlings, 2 followed a single needling, 2 followed linear extraction, and one followed trauma to an eye that had been successfully operated upon two years previously by linear extraction. In one instance the uveitis followed two unsuccessful attempts to remove the cataract through a corneal incision.

In case 48 the patient developed chronic uveitis, detachment of the retina, and vitreous hemorrhage after the second needling.

Sympathetic Ophthalmia

Sympathetic ophthalmia was diagnosed clinically and microscopically in case 36. Three months previously the patient had had a linear extraction accompanied by vitreous loss and a postoperative iris prolapse that was excised. Chronic uveitis supervened, and ten weeks after surgery the eye not operated upon developed uveitis. The exciting eye was enucleated.

Microscopic examination revealed a cyclitic membrane that included lens remnants and was invaded by plasma cells, lymphocytes, and macrophages. There was marked inflammation, granulomatous in nature, of the rest of the iris, ciliary body, and anterior choroid. The optic nerve was swollen and there were inflammatory cells about some of the smaller vessels. The picture was typical of sympathetic ophthalmia. Treatment of the remaining eye by cortisone therapy resulted in recovery with 20/30 vision.

In case 33, which was diagnosed clinically as endophthalmitis, there was diffuse infiltration of the choroid by lymphocytes and plasma cells suggestive of early sympathetic ophthalmia. It was impossible in this case to obtain any clinical information on the remaining eye.

Cyclitic Membrane Formation

Dense cyclitic membranes had formed in 12 cases: 6 after multiple needlings, 2 after a single discission, 2 after linear extraction, one after a Ziegler through-and-through operation, and one after an unknown type of surgery (fig. 9). In 8 of these cases the eye was enucleated between seven days and six months after surgery. In 4 cases enucleation took place eighteen, twenty-three, twenty-four and thirty-seven years after surgery. In case 14, in which the eye was enucleated twenty-three years after surgery, the cyclitic membrane contained bone, indicating that it had been present for many years. The other 3 cases (20, 47, and 50) were phthisical eyes, which also indicated that the cyclitic membrane was of long standing.

Pupillary Membrane Formation

Occlusion of the pupil by membrane formation occurred in 12 cases. In case 47, in which the type of surgery had not been recorded, there was occlusion of the pupil by a dense pupillary membrane which extended along the anterior surface of the iris. Anterior peripheral synechias were also present. While details of these cases are not available, examination of the slides shows a small pupil in each case and suggests that they were cases in which the pupil could not be dilated well before surgery. Clinically these cases always result in occlusion

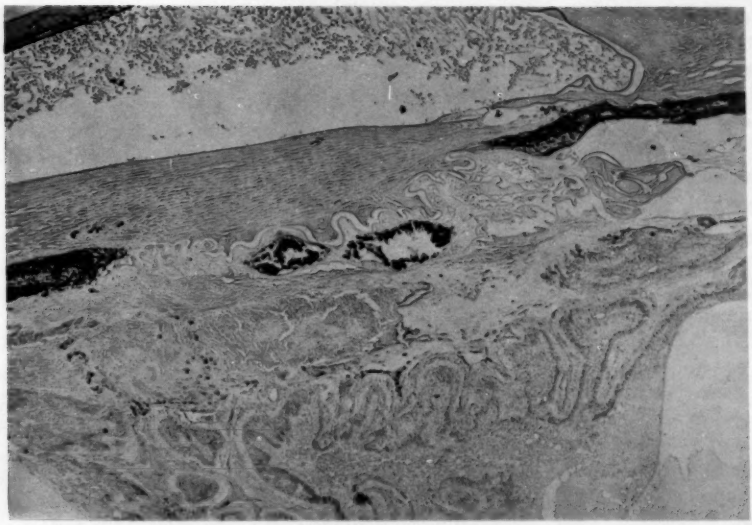

FIG. 9—Dense cyclitic membrane with lens capsule and retina incorporated.

of the pupil postoperatively. This had been recognized for many years. Von Graefe observed this and warned against needling congenital cataracts if the pupil could not be well dilated preoperatively.

Panophthalmitis

Panophthalmitis had obtained in 5 cases. Case 12 was probably a linear extraction followed by panophthalmitis and later phthisis bulbi. In case 13, multiple needlings had been followed by an attempt to remove the remainder of the lens through a limbal incision. Cases 23, 28, and 54 were linear extractions, followed within a few days by panophthalmitis. Thus, all 5 instances of panophthalmitis followed incision of the eyeball.

Phthisis Bulbi

Phthisis bulbi was found by Huerkamp[11] in 69 per cent of eyes that developed "severe complications." In this series of enucleated eyes, only 10 (17.9 per cent) were phthisical. In case 47 the phthisis bulbi followed some unrecorded operative complication. Cases 50 and 54 were linear extractions in which the phthisis followed a low-grade uveitis of long standing. One case (case 12) followed panophthalmitis after linear extraction; 4 followed severe inflammation after the last of multiple needlings that were done, respectively, thirteen, sixteen, eighteen, and twenty years before enucleation. In case 48 the phthisis resulted from recurrent hemorrhage and inflammation following trauma five months after the second needling.

Corneal Changes

Corneal changes were noted in 24 (42 per cent) of the series. These ranged from edema to chronic pannus, degenerative changes, and suppurative keratitis. In 16 of these cases there was also glaucoma, and in 8 of the 16 it was absolute. In 3 cases the keratitis appeared to be associated with vitreous in the anterior chamber and against the posterior cor-

neal surface. Bullous keratitis was seen in 3 cases. In view of Denig's observation of an apparent association between retinal detachment and corneal changes, it is interesting that 13 cases, or approximately 50 per cent of the cases in this series showing microscopic corneal changes, also showed retinal detachment (fig. 10).

neath the epithelium. In addition, there were degenerative changes in the corneal stroma, and the corneal endothelium was atrophic.

Some of the eyes with corneal changes warrant special consideration:

Case 19 is an example of lens capsule incarcerated in the operative wound af-

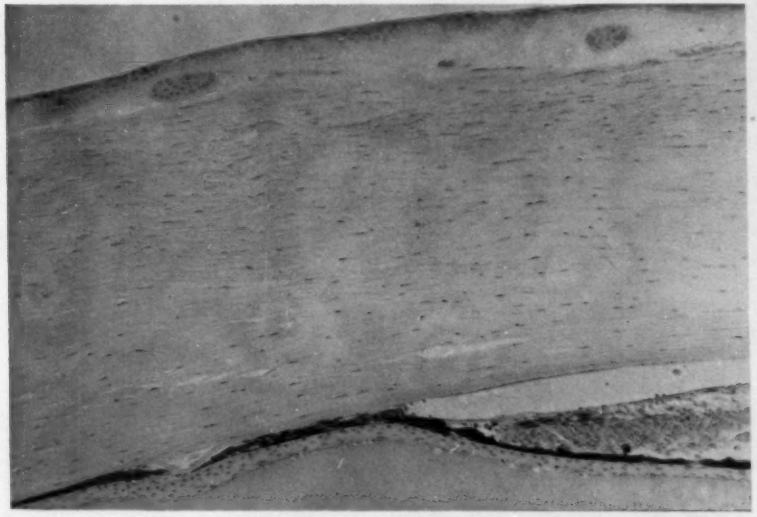

FIG. 10—Corneal changes associated with old detachment of the retina.

By far the commonest finding was corneal edema with chronic inflammation and vascularization. Case 35 illustrated this type of change. The patient, a 29-year-old man, had had multiple discissions when he was 4 years of age. Twenty-five years after the last operation the eye was enucleated because of pain. Vision had been lost for some time before surgery, apparently due to a detachment of the retina. The corneal epithelium was thin and irregular. Centrally, in the region where Bowman's membrane was interrupted, there was beginning epidermidalization of the epithelium and loose fibrinous tissue beter linear extraction. It emphasizes the importance of being certain that the corneal wound does not contain lens capsule, especially when an iridectomy has been done; when a round pupil operation has been performed, the iris is usually instrumental in preventing this complication.

In case 17, in which a diagnosis of sympathetic ophthalmia was made clinically but not confirmed microscopically, there was an unusual complication, namely, a purulent infection in the needling wound. Microscopic examination revealed purulent exudate, fibroblastic

proliferation, and hemorrhage filling the operative wound. The eye showed the changes of subacute iridocyclitis. This case emphasized the importance of observing meticulously the techniques of sterile surgery.

Case 9 had had a Ziegler through-and-through operation followed by glaucoma. A keratome incision was made in an attempt to remove the lens cortex. This was followed by an endophthalmitis. The scar of the incision was infiltrated with plasma cells.

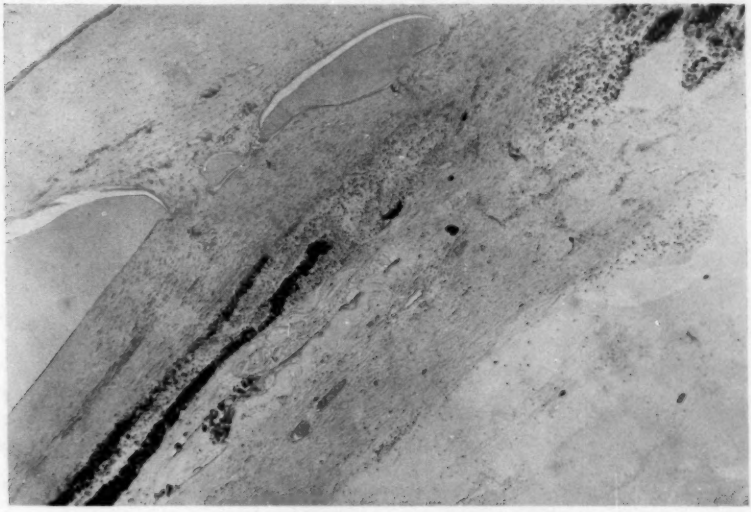

FIG. 11—Lens capsule adherent to wound.

Lens Remnants

Lens remnants were calcified in 4 cases, with no apparent relationship to the type of surgery. Lens capsule was adherent to iris or incarcerated into the cyclitic membrane in 5 cases: 2 after linear extraction, 2 after multiple needlings, and one after a Ziegler operation. In case 15 a small amount of lens capsule was adherent to the posterior corneal surface. In cases 19 and 33, both linear extraction cases, there was adherence of lens capsule to the corneal wound (fig. 11). In case 19 the scar tissue was attached to the lens capsule with the subcapsular epithelium on one side, and continued over the inner corneal surface to attach to lens remnants on the opposite side. These cases would seem to indicate the value of a large capsulotomy in linear extraction and to emphasize the importance of being certain that there are no lens remnants in the wound at the end of surgery. They also provide another argument in favor of round pupil operation if the pupil can be widely dilated.

Case 4 revealed a bulging scar which was the result of iridencleisis at the limbus. A fibrous membrane extended inward from the anterior edge of the ciliary body to lens remnants and replaced the chamber angle meshwork and the iris. In case 2 the lens capsule on one

side, with a small amount of degenerated substance, was attached to the iris, and lens epithelium grew onto the pupillary margin. In case 24 the lens was completely replaced by a large, thick mass of well-organized connective tissue to which iris, ciliary body, and retina adhered. A few capsular remnants were seen in the anterior part of this tissue. In case 49 there were posterior synechias to the lens capsule, and in case 55 lens remnants were adherent to the fibrovascular tissue occupying the anterior chamber.

Optic Nerve Changes

Optic atrophy was present in 32 cases, of which 23 had been subjected to multiple needlings. In 7 of the cases, or 31 per cent, there was a glaucomatous cup. In the cases of retinal detachment it was characteristic that the retina was pulled away from the disc. This was illustrated by case 8, in which there was gliosis of the nerve head, which was pulled forward and contained laminated and apparently calcific drusen. The lamina cribrosa did not appear to be depressed. In case 33 the retina was similarly detached and appeared to have been partially torn from the disc by the traction on the retina (fig. 12), although an artefact must be considered.

In case 11 the nerve head was detached from the deeply depressed lamina cribrosa and the retina was pulled into and over the disc, apparently by traction from retinal scars near the disc.

In case 41 the optic nerve showed deep cupping which was filled with proliferated glial tissue.

Ossification

Ossification was present in 5 eyes. In case 14, in which there had been multiple needlings twenty-three years before enucleation, there was ossification

FIG. 12—Detached retina showing traction on the disc.

in the cyclitic membrane and posterior choroid; in case 16, enucleated thirty-two years after multiple needlings, the ossification was in the posterior choroid; in case 43, enucleated fifty years after multiple needlings, and in case 50, in which a linear extraction had been done thirty-seven years before enucleation, there was also ossification in the choroid; while in case 46 the ossification occurred in the old detached retina. How long the retinal detachments had been present could not be determined. As was to be expected, ossification was present only in cases in which the pathologic changes had been of long standing.

Drusen

Drusen were seen rather frequently. The youngest patient in this group of cases was a girl 4 years of age, who had had a linear extraction three years prior to enucleation. The formation of drusen is a common finding after detachment of the retina.

Retinoblastoma

Case 25 warrants special consideration (fig. 13). The patient, who was 15 months old at the time of enucleation, had been seen six months previously. At that time he had an opaque lens of the right eye which, the mother stated, had been present for several months. The condition was diagnosed as infantile cataract, and a needling operation was performed. At the time of surgery the surgeon noted that the lens was "very opaque and semisolid, appearing more like calcium." Six months later the patient was again brought to the doctor; and at that time, the "whole anterior chamber seemed full of calcium-like material," and the eye was enucleated.

Microscopic examination of the enucleated eye revealed a retinoblastoma with necrosis and calcium degeneration (stage III). The tumor had invaded almost all of the structures of the eye. This case points up the importance of keeping retinoblastoma in mind, especially in those cases in which there is a history of the development of the opacity several months after birth.

Pseudotumor

Case 51 is of interest as a problem in differential diagnosis. A congenital cataract had been needled when the patient was 2 years of age. This was followed by the development of a pigmented tumor-like mass immediately behind the pupil. The mass failed to grow over a period of ten years, and the eye was then enucleated because of a chronic uveitis. Microscopic examination revealed retinal detachment with adhesion of the retina to lens remnants in the pupillary area, associated with proliferation of the pigmented epithelium of the ciliary body onto the anterior surface of the detached retina. These, then, were the components of the pigmented mass that had simulated a melanoma.

Spontaneous Expulsive Hemorrhage

So far as could be determined, spontaneous expulsive hemorrhage, though a rare complication of cataract extraction, has never been reported in connection with congenital cataract surgery. In case 53, a 6-year-old boy was admitted to the hospital for extracapsular extraction of a congenital cataract present from birth. Clinical examination revealed an opacity so diffuse that the fundus could not be visualized. Light perception and projection were present. Aside from the lens changes, the eye appeared to be normal. At surgery, when the lens capsule was grasped with lens capsule forceps, a massive intraocular expulsive hemorrhage occurred (fig. 14). Permission for enucleation was delayed one week. Microscopic examination revealed a massive detachment of the choroid by hemorrhage. The hemor-

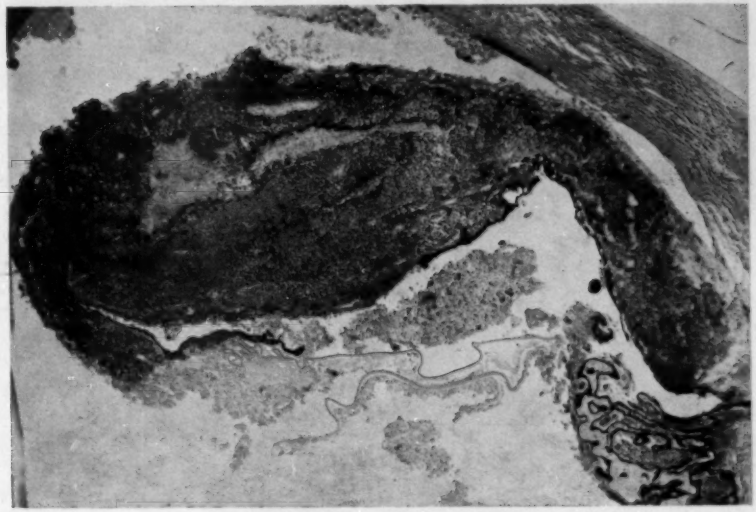

FIG. 13—Retinoblastoma filling anterior chamber.

rhage had ruptured the choroid anteriorly and was beneath the detached retina and in the vitreous. Aside from the changes resulting from the presence of the hemorrhage, no abnormalities were demonstrable during the week before enucleation was performed. This occurrence of spontaneous expulsive hemorrhage in a child at the time of cataract surgery would seem to be the only one on record.

AGE AT THE TIME OF SURGERY

In 11 (19 per cent) of the 56 eyes enucleated after congenital cataract surgery, the first operation was performed when the patient was one year old or younger; the youngest child was 3 months old. In 6 cases the only available history was that the first operation was "in childhood." In the remainder of the patients, surgery was performed between one and 30 years of age. It should be borne in mind that those cases in which surgery is indicated very early are also the cases with the greatest number of other congenital anomalies. It is difficult to draw any conclusions from this group of cases as to the influence of age on the complications of congenital cataract surgery.

SUMMARY AND CONCLUSIONS

In many of these cases, the history and clinical data were so meager that it was impossible to correlate accurately the clinical findings with the pathologic changes. Attempts to obtain further data were in most instances unsuccessful. For this reason, conclusions or opinions are to a certain extent based on supposition.

Fifty-six (56) eyes enucleated after congenital cataract surgery were subjected to histopathologic study. Needlings had been performed in 71.6 per cent of the 56 eyes; and in 72 per cent of the needlings, multiple discissions had been done. In one instance, a Ziegler through-and-through operation had been performed.

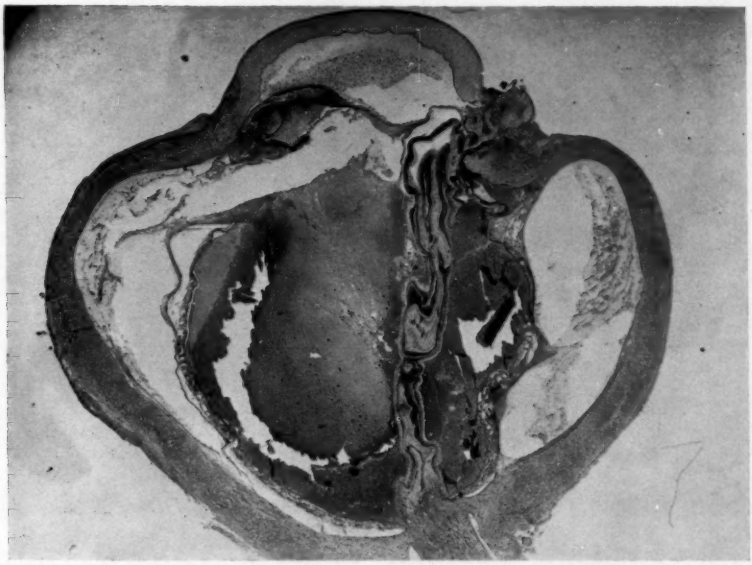

FIG. 14—Expulsive hemorrhage occurring on operating table in 6-year-old patient.

TABLE VI

PRINCIPAL COMPLICATIONS OBSERVED IN 56 EYES REMOVED AFTER FAILURE OF CONGENITAL CATARACT SURGERY

	DISCISSION		LINEAR EXTRACTION	ZIEGLER OPERATION	UNKNOWN TYPE	NUMBER	PERCENTAGE TOTAL CASES
	SINGLE	MULTIPLE					
Glaucoma	5 8.9%	16 28.1%	4 7.1%	1 1.9%		26	46
Retinal detachment	5 8.9%	13 23.1%	4 7.1%		1 1.9%	23	41
Uveitis	2 3.5%	7 12.5%	2 3.5%	1.9%	1 1.9%	13	23.3
Sympathetic ophthalmia			1 1.9%			1	1.9
Cyclitic membrane	2 3.5%	6 10.6%	2 3.5%	1.9%	1 1.9%	12	21.4
Panophthalmitis			4 7.1%		1 1.9%	5	9
Phthisis bulbi		6 10.6%	3 5.4%		1 1.9%	10	17.9
Optic atrophy	6 10.7%	19 33.9%	5 8.9%		2 3.5%	32	57

Glaucoma was present in 26 (46 per cent) of the 56 eyes; and in 75 per cent of these, the glaucoma followed a needling or, more often, multiple needlings. Uveitis, intraocular hemorrhage, the formation of cyclitic membrane, and vitreous block were contributing factors. The case of vitreous block of the pupil occurred after a second needling and illustrates one of the complications that can occur in cases in which the pupil cannot be dilated preoperatively. When this is the case, the vitreous can come forward and block the pupil. In the case in which a Ziegler operation was done, glaucoma developed as a result of the swelling of the cortex. When attempts were made to wash out the anterior chamber, which contained vitreous, complications arose that resulted in endophthalmitis. While no conclusions can be drawn from a single case, this outcome confirms what has been observed clinically, namely, that an attempt to evacuate lens cortex in the presence of vitreous in the anterior chamber usually ends in disaster.

The frequency with which anterior peripheral synechias were observed seems to emphasize the importance of early restoration of the chamber and to substantiate the clinical observation that the injection of air at the conclusion of the operation is indicated, especially in those cases in which the anterior chamber has been opened.

Detachment of the retina occurred in 23 (41 per cent) of the cases in this series. In 18 instances it followed needling. Of particular interest was the fact that in 37.5 per cent of the cases of multiple needling, detachment of the retina occurred in from fifteen to thirty-three years, or an average of 22.2 years, after surgery. The theory of Shapland and others that traction is a factor in the production of detachment would seem to be substantiated by 6 cases in which a membrane formed between the anterior chamber and the retina, all in eyes that had had multiple discissions. The study of the eyes with delayed detachment of the retina apparently bears out the clinical observation that the more the vitreous is disturbed, the greater the chance of late detachment. Thus the operation of choice in congenital cataract, whenever possible, should be some form of linear extraction or some form of surgery in which the posterior capsule and the vitreous face are not disturbed.

Pupillary membrane formation was present in 12 cases. While it was not possible to obtain detailed data in these cases, they may very well have been cases in which it was not possible to dilate the pupil well before surgery, since in most of them the pupillary space was small. This would point to the advisability of doing a full iridectomy in those cases in which it is not possible to dilate the pupil widely before surgery.

Panophthalmitis occurred in 5 instances, in all of which the eyeball had been opened, either for a linear extraction or for an iridectomy for glaucoma, after multiple discissions. It must be borne in mind that linear extraction does carry a greater risk of intraocular infection than discission.

Corneal changes ranging from edema to chronic pannus, degenerative changes, and suppurative keratitis, occurred in 24 (42 per cent) of the series. In 50 per cent of these, retinal detachment occurred.

In this series, only 16 per cent showed phthisis bulbi in contrast to the 69 per cent reported by Huerkamp from clinical observations. Perhaps this was due to the fact that phthisis usually causes no symptoms, and therefore the eye is often left alone.

The adherence of lens capsule to the wound in 7 instances emphasizes the

necessity of being certain that there are no capsular remnants in the wound at the completion of the operation.

It is noteworthy that in only one instance was it possible to establish the diagnosis of sympathetic ophthalmia. In a second patient the examination suggested the possibility of early sympathetic ophthalmia, but there were insufficient data to warrant a diagnosis.

The presence of retinoblastoma in one case as a contributing factor to the opacification of the lens emphasizes the importance of keeping this condition in mind, especially in those cases in which the cataract develops after birth and in which the fundus cannot be visualized even grossly.

From the data available in this series, it was not possible to correlate the incidence or nature of the postoperative complications with the age of the patient when first operated upon.

The following conclusions of practical value may be drawn from this study:

1. Linear extraction is the safest operation for congenital cataract so far as both immediate results and avoidance of late complications are concerned.

2. In those patients in whom it is impossible to dilate the pupil well before surgery, a full iridectomy should be performed.

3. In cases of aftercataract, membranous cataract, or other cataract in which linear extraction is contraindicated, the procedure chosen should be the one that will do the least possible damage to the vitreous in order to avoid late detachment of the retina.

4. Multiple needlings seem to be the least desirable of all procedures, since our statisics show that 37.9 per cent of the cases subjected to multiple needlings developed late detachment of the retina on an average of 22.2 years after the last needling.

5. While this series of late detachments is small, statistically the occurrence of late retinal detachments following linear extraction is significantly less than the number of late retinal detachments following multiple discissions.

I am indebted to Dr. Frederick Carriker and Mr. Ben Goldfeller for the photomicrography which, because of the faded conditions of some of the specimens, was extremely difficult.

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NEW DRUGS AND TECHNIQUES FOR OCULAR ANESTHESIA

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IT is my purpose to summarize some recent developments in the field of local anesthesia as applied to ophthalmology. A number of new local anesthetics are available, and others are being investigated. There is a better understanding of the mechanism of action of such adjuncts as hyaluronidase and vasoconstrictors; also, new knowledge has been obtained concerning the distribution and effects of drugs following injections around the globe. Finally, several new techniques of injection have been reported.

NEW LOCAL ANESTHETICS

In recent years, a number of promising new local anesthetics have been developed. In the evaluation of these drugs it should be kept in mind that a diversity of factors determine the usefulness of a given drug in ophthalmology (table I). For example, a drug to be useful as a topical anesthetic must penetrate the epithelium readily and be relatively nonirritating. When all factors are considered, it seems unlikely that there can be a single local anesthetic superior to others for all phases of ophthalmic practice. For this reason, most recent research has been directed toward finding the most suitable agents for specific purposes rather than toward the development of all-purpose drugs.

TABLE I

FACTORS DETERMINING USEFULNESS OF LOCAL ANESTHETICS IN OPHTHALMOLOGY

1. Epithelial penetration
2. Anesthetic potency
3. Duration of effects
4. Secondary effects, e.g., mydriasis
5. Systemic toxicity
6. Tissue irritation
7. Allergenicity
8. Sterility of solutions
9. Chemical stability in water

An important need in the daily office practice of ophthalmology has been for stable, nonirritating, short-acting drugs which would rapidly induce corneal and conjunctival anesthesia without undesirable side effects, such as mydriasis. For this purpose, 0.5 per cent tetracaine hydrochloride (pontocaine) in aqueous solution has been most commonly used by American ophthalmologists. It is effective, but its solutions are rather irritating and frequently induce epithelial changes. Perhaps the greatest disadvantage of tetracaine has been the occasional development of hypersensitivity reactions to the drug. The incidence of hypersensitivity seems to vary in different sections, apparently being higher in areas where local anesthetics are prescribed for prolonged home use. Three new compounds seem to have advantages over tetracaine for office procedures such as tonometry and gonioscopy, and for short operative procedures.

One of these new compounds, benoxinate (dorsacaine), is closely related chemically to procaine and tetracaine (fig. 1). Dorsacaine has been studied at the University of Oregon Medical School since 1952.[14] Its hydrochloride

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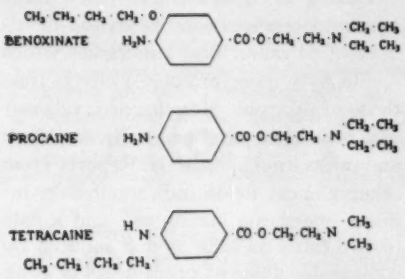

FIG. 1—Benoxinate, tetracaine and procaine are similar in chemical structure but differ in physical properties and pharmacologic effects.

salt is a white powder that is readily soluble in water, forming clear stable solutions which can be sterilized by boiling. It is commonly used in 0.4 per cent concentration in aqueous solutions made physiologically isotonic with the addition of sodium chloride. Solutions of this concentration have an anesthetic potency comparable to 0.5 per cent tetracaine hydrochloride in the onset, intensity, and duration of the anesthetic effects, but benoxinate has several advantages. If preservatives are not added, the isotonic solutions produce little or no irritation in uninflamed eyes. Disturbances in the corneal epithelium or hyperemia of the conjunctiva seldom are noted by slit lamp biomicroscopy after even two or three instillations, whereas these changes seem quite consistent after repeated instillations of 0.5 per cent tetracaine hydrochloride. For tonometry, a single instillation of 0.4 per cent benoxinate generally is adequate. If the drop is not well instilled or is squeezed out by blepharospasm, a second drop invariably permits a measurement of the ocular tension. When an imbedded foreign body is to be removed from the cornea, three instillations at 90-second intervals usually ensure adequate surface anesthesia within four to five minutes. Solutions of 0.5 per cent benoxinate prepared in sterile water do not seem to become contaminated by bacteria, perhaps because of the relatively low surface tension of these solutions.

Two other new compounds which rate highly as topical anesthetics for office use are dyclonine hydrochloride (dyclone) and ophthaine.[1,4] Like benoxinate, both form stable aqueous solutions. They are used in concentrations of 0.5 per cent. These two compounds are chemically similar (fig. 2), but belong

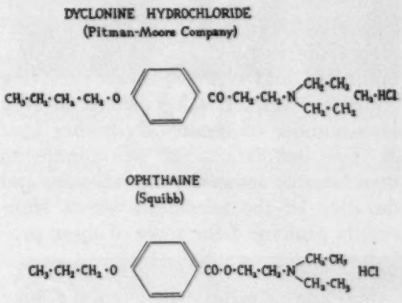

FIG. 2—Dyclone and ophthaine are similar in chemical structure, physical properties and pharmacologic effects. In 0.5 per cent aqueous solution, they rapidly induce corneal and conjunctival anesthesia with minimal irritation.

to a different chemical group than benoxinate, tetracaine, and procaine; therefore, they can be used in patients who might have acquired hypersensitivity to any of that group. Both dyclonine and ophthaine have the advantage over tetracaine of rapidly producing surface anesthesia of relatively short duration with a minimum of such side effects as changes in the corneal epithelium. Like benoxinate, both of these compounds seem to have some antimicrobial properties. In vitro studies indicate that dyclonine hydrochloride possesses both bactericidal and fungicidal properties. It is highly active against Staphylococcus aureus, a common pathogen growing along the lid margins. The phenol coefficient of dyclonine in aqueous solution has been reported to be 3.6.[5]

For cataract surgery, many American ophthalmic surgeons still prefer 4 to 5 per cent cocaine instillations to stronger concentrations of the shorter-acting topical anesthetics. For cataract extraction, cocaine has the advantages of producing deep anesthesia, increasing mydriasis, and enhancing the vasoconstrictor effects of epinephrine. Hypersensitivity reactions to cocaine are extremely rare. In recent years, many surgeons have been using stronger concentrations or repeated instillations of the shorter-acting anesthetics. For example, Atkinson advocates one per cent tetracaine.[2] Repeated instillations of dorsacaine, ophthaine, and dyclone also are receiving trials for cataract and glaucoma surgery in a number of clinics. By giving four or five instillations at two-minute to three-minute intervals, the intensity and duration of the anesthesia seems sufficiently prolonged for some of these procedures.

Procaine remains the most widely used anesthetic for injection, but there has been need for an alternative agent for patients who have developed hypersensitivity to it. In the postwar period, xylocaine (lidocaine) has received increasing recognition not only as an alternative agent but also as a drug comparable to procaine in effectiveness, tissue tolerance, and relatively low toxicity. It has been said that xylocaine is more potent, diffuses more rapidly, and has more lasting effects than comparable doses of procaine; however, these claims have not been universally accepted. In fact, Scheie and his associates found that the duration of retrobulbar anesthesia in rabbits was less prolonged than that induced by procaine.[12] In the higher concentrations, xylocaine also seems to be more irritating to the tissues, but Russell and Guyton found 4 per cent aqueous solutions to be well tolerated and highly effective for akinesia and retrobulbar injections.[11]

During the past several years, there has been renewed interest in the development of other local anesthetics which would have more prolonged effects than those of procaine. Hexylcaine (cyclaine) is a new agent used primarily for block and infiltration anesthesia. Reports from other clinical fields indicate that it induces anesthesia lasting one and a half to two times as long as that induced by comparable doses of procaine.[3] The drug has received little use in ophthalmology. Scheie and his co-workers have reported on still another new compound, U-0045.[12] They observed that even in dilute concentration this agent had anesthetic effects lasting as long as eight to twelve hours in the rabbit. A combination of drugs to produce long-lasting anesthesia, marketed under the trade name of efocaine, was introduced several years ago. This preparation is too irritating to inject around the globe.

TECHNIQUES OF INJECTION ANESTHESIA

Important developments are occurring in relation to the technique of injection anesthesia for ophthalmic surgery. In the past, all injections behind the globe have been spoken of as retrobulbar injections, but it has become increasingly evident that the effects vary markedly with the site and depth of the injection, with the concentration and quantity of the anesthetic solution, and with the nature of the added adjuvant. The possible effects of retrobulbar injection are motor, sensory, and autonomic (table II). Earlier authors wrote of ciliary ganglion block as the major aim of retrobulbar injection; now it is recognized that ocular anesthesia is not solely dependent upon injecting the drug solution into the region of the ciliary ganglion. Ocular anesthesia can be induced by blocking the main sensory branches near the apex of the orbit or the ciliary nerves behind the globe.

TABLE II

POSSIBLE EFFECTS OF RETROBULBAR INJECTIONS OF LOCAL ANESTHETICS

1. Motor: paresis of extraocular muscles
2. Sensory: anesthesia, impaired vision
3. Autonomic: hypotension, pupillary changes, vasodilatation, and others

For many operations, akinesia of the extraocular muscles is an added objective of retrobulbar injection. Gifford has reviewed the distribution of the motor nerves in the orbit and has found that a deep orbital injection is most productive of a high percentage of oculomotor blocking.[8] He uses a 2-inch needle and makes the injection laterally and deep into the orbit. Damage to the optic nerve and the incidence of hemorrhage are minimized with the use of a blunt-tip needle. O'Brien, in his teachings, designated deep orbital injections directed toward the superior orbital fissure as "apical" injections.[9,10] O'Brien limited the use of the term "retrobulbar" to the small (1.0 to 1.5 cc.) injections confined to the relatively avascular region just behind the globe but temporal to the optic nerve. In contrast to the apical region, there are no motor nerves or large vessels in this area; consequently, motor effects are absent or minimal, and retrobulbar hemorrhage rarely occurs. This "anterior" retrobulbar region easily can be reached with a 1-inch needle via the inferior lateral fornix (fig. 3). The respective locations of 1-inch and 2-inch needles in the orbit are shown in a coronal section through the orbit (fig. 4).

The anterior type of retrobulbar injection is directed toward blocking only the ciliary nerves; therefore, it produces only sensory and autonomic effects. Adequate ocular anesthesia without oculomotor paresis can be obtained by the injection of only 1.0 to 1.5 cc. of 2 to 4 per cent procaine directly behind the eye. The distinction between apical injection and injection just behind the globe seems particularly important in re-

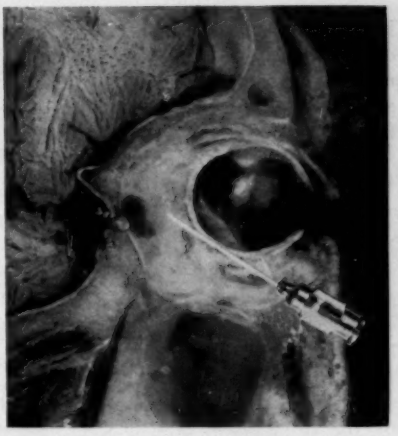

FIG. 3—Sagittal section through the right orbit temporal to the optic nerve. The ciliary nerves behind the globe can be reached with a one-inch needle directed upward and nasally from the inferior temporal fornix. There are no large blood vessels or nerves to the extraocular muscles in the injection area.

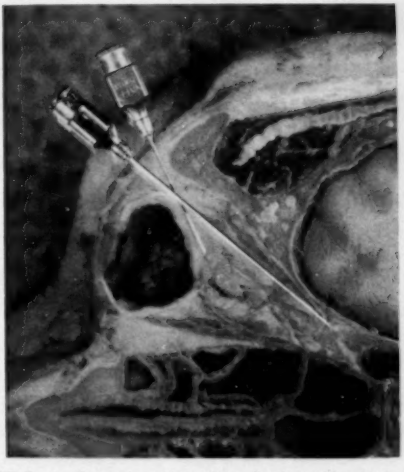

FIG. 4—Coronal section through the orbit at the level of the medial and lateral rectus muscles. Relative position of the needles in anterior retrobular (one-inch needle) and apical (two-inch needle) injections made through the lower lid.

lation to alcohol injections for the relief of pain. Then the injection should be made anteriorly in the orbit to produce a sensory block without creating disfiguring oculomotor paralysis. By the same logic, apical injections of alcohol should be avoided.

The most commonly used retrobulbar injection is intermediate in depth between the apical injection of Gifford and the anterior retrobulbar injection of O'Brien. It has been advocated by Atkinson and others.[2] Atkinson has designated this type of retrobulbar injection as a "cone" injection because the bulk of the drug solution is placed in the muscle cone about one centimeter behind the globe. This is in the region of the ciliary ganglion. The large vessels at the apex of the orbit are avoided. The incidence of retrobulbar hemorrhage is less than with apical injections.

Using a one-and-a-half-inch needle, Atkinson injects 2 to 3 cc. of anesthetic solution containing hyaluronidase into the cone. The addition of hyaluronidase permits the larger volume of drug solution to be used because the enzyme facilitates rapid diffusion in the tissues. Also, it increases the motor effects of the injection by enhanced penetration of the nerve sheaths. If this intermediate depth of injection is selected and 2 to 3 cc. of four per cent procaine are introduced with epinephrine and hyaluronidase* added, deep anesthesia, oculomotor paresis, and some reduction in ocular tension can be expected. Duration of the anesthesia generally will be from 50 to 100 minutes. It probably will be shorter if hyaluronidase is added.

The inferior temporal quadrant of the orbit generally is selected for retrobulbar injections. If the injection is made through the skin of the lower lid, the needle reaches the orbit via the orbital septum. If the injection is made through the conjunctiva, the needle passes through the connective tissue of the fornix. In either case, the needle enters the muscle cone outside of and behind Tenon's capsule. Another technique is to inject under Tenon's capsule just behind the equator of the globe. Then the anesthetic drug solution spreads between Tenon's capsule and the sclera to reach the ciliary nerves in the retrobulbar region. Anesthesia and autonomic effects such as pupillary changes and hypotony occur. We have designated this as a "posterior subcapsular injection." Anterior subcapsular injections have different effects and will be discussed later.

At the University of Oregon Medical School, the following technique of posterior subcapsular injection is used occasionally prior to some cataract and glaucoma operations. The conjunctiva and Tenon's capsule are picked up with forceps in the equatorial region of the superior temporal quadrant. Then the needle is passed posteriorly between Tenon's capsule and the globe to permit injections both in the retrobulbar area and under the belly of the superior rectus muscle (fig. 5). This type of posterior subcapsular injection produces anesthesia and an effective superior rectus block. It is a useful procedure when it is desirable to obtain good anesthesia and to block only the superior rectus. A bridle suture then is unnecessary to rotate the eye downward. I do not use the posterior subcapsular technique routinely for cataract surgery. For example, in patients with a sunken eye and a prominent brow, the superior orbital margin may make the superior rectus block difficult. Then, in some cases I prefer a more complete oculomotor block. The posterior subcapsular technique can be applied to any quadrant. The technique is particularly suitable

*Hyaluronidase generally is used in a concentration of 8 to 10 turbidity reducing units per cubic centimeter of anesthetic solution.

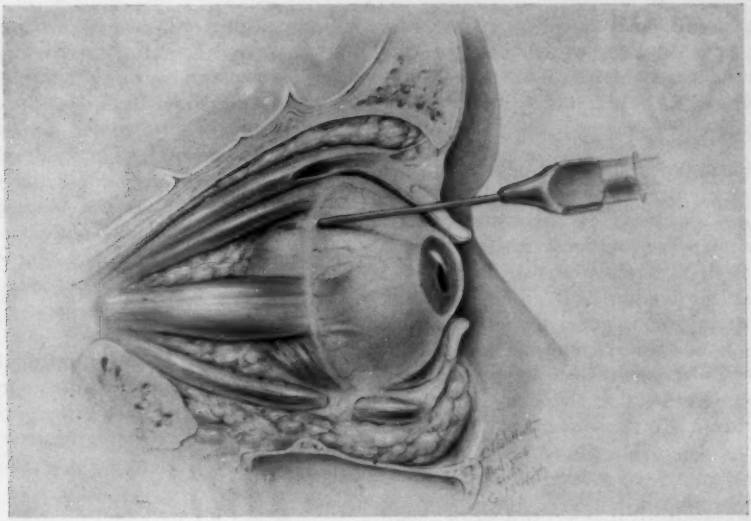

FIG. 5—Posterior subcapsular injection. The needle is passed through the bulbar conjunctiva and Tenon's capsule in the superior temporal quadrant near the fornix. The drug solution is injected posteriorly between the capsule and the sclera to block the ciliary nerves. Then the needle can be passed posteriorly to block the superior rectus.

for operations for retinal detachment or other procedures requiring selective blocking of motor nerves.

It should be pointed out that anesthesia and akinesia of the extraocular muscles for strabismus surgery can be effectively obtained by blocking the nerves. Attempts to inject directly into the muscle belly, as has been advocated in older textbooks, may injure the muscle and result in a higher incidence of hemorrhage.

It long has been recognized that retrobulbar injections of procaine and epinephrine lower ocular tension in some eyes. Recently, it has been demonsrated by de Roetth and his co-workers[7] that the hypotensive effects of retrobulbar injections can result from the procaine and are not due entirely to the added epinephrine, as postulated by some earlier authors. From tonographic studies, de Roetth and his associates concluded that retrobulbar procaine reduced aqueous inflow in some eyes with chronic glaucoma. The hypotensive effects of procaine are enhanced by the addition of hyaluronidase, apparently because of the greater diffusion of procaine. Epinephrine results in better retention of the procaine in the orbit, but in addition it possibly may have some hypotensive action. External pressure on the globe also lowers ocular tension.

It has been known for years that some patients have marked impairment of vision following retrobulbar injections of local anesthetics. This loss of vision, although transitory, is alarming to patients. Recently, Carroll and de Roetth have made a systematic study and have found that some impairment of vision occurred in a high percentage of patients, particularly when concentrations as high as 4 to 8 per cent procaine were injected into the muscle cone with a 35

mm. needle.[6] Recovery of vision was invariable, so it was evident that the loss of vision was due to the effect of procaine on the optic nerve rather than trauma to the nerve by the needle.

Subconjunctival injections of drugs commonly are made near the limbus to induce anesthesia of the anterior segment; however, little or no distinction has been made between injections just under the conjunctival epithelium and deeper injections under the anterior extension of Tenon's capsule. My associates and I are conducting experiments which indicate that there are clinically significant differences in the penetration of drugs into the anterior segment from the two tissue levels.[15]

Subconjunctival injections made just under the epithelium but external to Tenon's capsule have been designated as subepithelial injections. Schlegel and I have found that from this level Tenon's capsule acts as a considerable barrier to the passage of drugs into the cornea and iris.[13] Also, the conjunctival vessels, unless constricted with epinephrine, rapidly carry the drug into the general circulation. In contrast, injections made under Tenon's capsule are brought into direct contact with the limbic stroma and sclera. To induce anesthesia of the iris, anterior subcapsular injections seem considerably more effective than subepithelial injections. In fact, quantitative tissue analyses indicate that following the subepithelial injection of one per cent procaine, concentration of the drug in the iris reaches only minimal levels for anesthesia.[13] The addition of hyaluronidase did not enhance ocular penetration of the anesthetic, but epinephrine increased the effectiveness of the injection.

The relative effectiveness of the two levels of subconjunctival injections can be demonstrated with injections of 1:1000 epinephrine hydrochloride in aqueous solution. This drug produces a segmental contraction of the iris dilator fibers. If comparable doses are injected under the epithelium and under the capsule at different sites in the same eye, dilatation of the pupil generally develops more rapidly near the site of the subcapsular injection.* At the University of Oregon Medical School, anterior subcapsular injections of 2 per cent procaine have been used to produce iris anesthesia for glaucoma surgery. Good anesthesia generally is obtained, and the mydriasis and hypotony of retrobulbar injections are avoided. With marked hypotony and loss of miosis, the peripheral iris is less apt to prolapse into the wound; then the iridectomy is more difficult to make.

SUMMARY

Three new short-acting drugs seem highly effective for the rapid induction of topical anesthesia of short duration with minimal side effects. These compounds seem to have advantages over tetracaine for routine office practice and short operative procedures. Of the new agents, benoxinate is chemically related to tetracaine and procaine. The other two drugs, dyclonine and ophthaine, differ chemically from the older drugs and can be used in patients who have become hypersensitive to the procaine derivatives.

For injections around the eye, xylocaine has been found to be an effective substitute for procaine. The two drugs have comparable effectiveness and toxicity. Hexylcaine merits investigation.

The motor, sensory, and autonomic effects of retrobulbar injections of anesthetic solutions have been clarified. A distinction has been made between the

*This was demonstrated in the motion picture Ocular Penetration of Drugs Following Subconjunctival and Subcapsular Injections, by Drs. Kenneth C. Swan, Paul Bailey, Jr., and Homer R. Crisman.

effects of various depths of retrobulbar injections. Injections near the apex of the orbit induce a relatively high percentage of motor block, but the large vessels in this region increase the risk of hemorrhage. "Cone injections" in the region of the ciliary ganglion produce adequate anesthesia, and if larger quantities of the drug solution are used and hyaluronidase is added to facilitate diffusion, motor blocking also results. Injections may also be made into the relatively avascular area directly behind the globe. There is little risk of hemorrhage, but this type of anterior retrobulbar injection, generally made with small amounts of drug solution, produces only sensory and autonomic effects. It is particularly useful for alcohol injections for relief of pain. Disfiguring deviations are avoided.

Still another technique of directing drugs to the retrobulbar area is to inject posteriorly under Tenon's capsule near the superior temporal equator. The drug solution follows Tenon's capsule back to the region of the ciliary nerves. In most patients, this posterior subcapsular technique also permits injection under the superior or other rectus muscles to block selectively the nerves to individual muscles.

Subconjunctival injections can be made subepithelially, that is, between the conjunctival epithelium and the forward extension of Tenon's capsule, or they can be made deeper between Tenon's capsule and the sclera. Subepithelial injections seem less effective than the anterior subcapsular injections in inducing the penetration of drugs into the iris and anterior segment. Subcapsular, rather than subepithelial, subconjunctival injections are recommended for anterior segment anesthesia.

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OBSERVATIONS ON ANESTHESIA FOR OCULAR SURGERY

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THE purpose of this paper is to summarize briefly the reports from a cross section of ophthalmologists in the United States on the anesthesia used for ocular surgery and to determine the safest and best procedure. It was a surprise to find such a large variety of drugs and methods used in the preparation of the patient and in the production of anesthesia and akinesia. However, with the majority of ophthalmologists the procedure is quite uniform. There are only a scattered few who employ the less commonly used drugs and procedures, some of which it might be well for more of us to adopt.

Over thirty drugs were reported as being used to produce sedation and analgesia. Ten different preparations are given to prevent nausea and vomiting. Twenty different anesthetic agents, in various strengths and combinations, are used to produce anesthesia and akinesia. Although the methods of preparing the patients and ways of producing anesthesia and akinesia vary, it is particularly striking that the majority of surgeons with whom I communicated appear to be well satisfied with their procedure and the results obtained. The data compiled from these responses indicate that there are many ways to reach the desired goal. However, it should be noted that the goal is apparently not the same with all ophthalmologists. Some prefer profound sedation; others, little or none. All appear to obtain good anesthesia, but many apparently do not strive for complete akinesia of the rectus muscles. A few prefer general anesthesia or narcosis.

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The importance of careful psychologic preparation of the patient and preanesthetic medication was emphasized by many. I am convinced that without careful preanesthetic preparation, good anesthesia and akinesia are rarely obtained regardless of the anesthetic agent used. Of the thirty different drugs reported as being used preoperatively to produce sedation and analgesia, by far the most popular were pentobarbital sodium (nembutal) and meperidrine hydrochloride (demerol). Secobarbital sodium (seconal) was next in favor. Chloral hydrate was the only other sedative used by any appreciable number.

With the wound firmly sutured, vomiting is rarely harmful and is no longer a dreaded occurrence following intraocular surgery, particularly cataract extraction. It is, however, undesirable and unpleasant. Of the ten different drugs reported as being used to prevent or stop nausea and vomiting, chlorpromazine hydrochloride (thorazine) is the one most frequently mentioned, and dimenhydrinate (dramamine) is next in favor. A few ophthalmologists and anesthesiologists are opposed to the use of thorazine and registered strong disapproval. A third drug, phenergan hydrochloride, has in my experience proved satisfactory. In a series of 200 consecutive cases, phenergan hydrochloride was used in the first hundred and chloropromazine hydrochloride (thorazine) in the second hundred. Otherwise the preparation and anesthetic were the same. Two of the patients were given phenergan vomited, and three who were given thorazine vomited also. However, two patients who received phenergan for their first cataract ex-

traction had no nausea or vomiting, whereas for the extraction on their second eye they received thorazine and had severe vomiting. In this series of 200, the eyes of the five patients who vomited were not harmed.

Careful preanesthetic preparation and complete anesthesia with the elimination of postoperative pain are good insurance against nausea and vomiting.

Most intraocular operations on adults can be done safely and painlessly with local anesthesia. Of the 454 ophthalmologists with whom I communicated, 93.2 per cent use local anesthesia; 6.8 per cent prefer to have the patient unconscious, and for this purpose, thiopental sodium (pentothal sodium) is the agent usually chosen. The preference was frequently qualified by the statement, "provided a competent anesthesiologist is available." Three reasons were given for preferring narcosis for ocular operations:

1. The surgeons felt more at ease, relaxed and less nervous concerning the operations with the patient unconscious.
2. The surgeons had become accustomed to it while in the Armed Forces.
3. Good anesthesia and akinesia had not been obtained with local anesthetics.

Many who use local anesthesia expressed themselves as feeling relaxed and at ease when employing that form, whereas the added risk of general anesthesia or narcosis, particularly with the elderly patient, worried them. The use of general narcosis for intraocular operations is probably more acceptable as one becomes accustomed to it and as efficient teamwork is developed between the ophthalmologist and anesthesiologist. Both methods certainly have their place in intraocular surgery.

For topical anesthesia, tetracaine hydrochloride (pontocaine) is the most popular anesthetic agent among the ten which were reported as being used. It was a surprise to learn that cocaine is still used by nearly 10 per cent of the ophthalmologists who answered my inquiry. The other eight topical anesthetic agents mentioned are used by only a scattered few. Benoxinate (dorsacaine), developed in Switzerland and known abroad as novesin, has been reported by Schlegel and Swan[6] to be an excellent topical anesthetic for minor operations and tonometry, but it is of too short duration for major ocular surgery. However, if instillations are repeated during the operation, anesthesia can be maintained. It is a good substitute if the patient is sensitive to tetracaine hydrochloride.

Procaine hydrochloride is the most popular anesthetic agent used for injection among those who said that they use local anesthesia to produce anesthesia and akinesia. The strength of procaine hydrochloride used varies from 0.5 per cent to 10 per cent. Since complete anesthesia and akinesia can be obtained with a 2 per cent solution, it does not seem wise to subject the patient to the added risk of having a toxic reaction by using a stronger solution.

Next in popularity to procaine hydrochloride as an anesthetic agent for injection is lidocaine hydrochloride (xylocaine). Its chief advantage over procaine hydrochloride is that its action is of longer duration, which minimizes postoperative pain. It diffuses more readily than procaine hydrochloride, thus producing more complete anesthesia and akinesia. The toxicity of xylocaine is about the same as procaine. The usual strength of xylocaine used is 2 per cent, although a few ophthalmologists use 1 per cent and several reported using a 4 per cent solution.

With a few exceptions, all with whom I communicated add epinephrine hydrochloride to the anesthetic solution, unless contraindicated. The advantages ascribed to the addition of epinephrine are that it retards systemic absorption of the anesthetic and hastens, intensifies and prolongs the anesthesia and akinesia.

Seventy-five per cent of those who prefer local anesthesia mentioned combining hyaluronidase with the anesthetic solution to enhance diffusion. Several remarked that in their opinion the addition of hyaluronidase to the retrobulbar injection has done more than any other thing in recent years to prevent complications, particularly vitreous loss during intraocular surgery.

Some ophthalmologists expressed an objection to the use of hyaluronidase in the anesthetic solution for retrobulbar injections, stating that the marked hypotony which occurred following its use made the cataract extraction more difficult. Dunnington and Chandler,[2] and others who have modified their technique of cataract extraction to take advantage of the hypotony, say "the softer, the better," which expresses my feelings about it. Vitreous loss and other complications are certainly less likely to occur with a soft eye. A second objection to the use of hyaluronidase, which was expressed by three surgeons, was that it shortened the duration of anesthesia and akinesia. This has not been my experience when epinephrine hydrochloride has been added to the anesthetic solution.

Akinesia of Orbicularis

To produce akinesia of the orbicularis, the Van Lint procedure and O'Brien modification were the two methods most frequently used. The Van Lint procedure was a two-to-one favorite over the O'Brien. A few ophthalmologists stated that they routinely used both. As Verhoeff might say, most ophthalmologists seem to have overlooked the simple and sure way of producing akinesia of the orbicularis, which I reported two years ago.[1] This injection, now slightly modified from the original report, is begun at the inferior edge of the zygomatic bone in line with the lateral margin of the orbit. The anesthetic solution is injected as the needle advances upward, across and close to the zygomatic bone at an angle of about 30 degrees from the temporal margin of the orbit (fig. 1). This injection is followed by *firm pressure* over the area of injection. A few ophthalmologists stated that they use 10 to 15 cubic centimeters of the anesthetic solution to produce akinesia of the orbicularis. With the zygomatic injection just described, this can be accomplished by the injection of 3 to 4 cubic centimeters.

Strong contractions of the orbicularis during convalescence undoubtedly interfere with wound healing and are responsible for many shallow chambers, but not all, as was shown when prolonged akinesia of the orbicularis was produced in a small series of patients for cataract extraction. In one patient the orbicularis remained inactive for three weeks and did not return to normal for two months; yet a shallow chamber occurred on the fourteenth day and remained shallow for five days.

Unfortunately, no safe anesthetic agent has been developed that will produce akinesia of the rectus muscles of sufficient duration to eliminate those muscles as a factor in the production of a shallow or flat chamber, which so often occurs after the tenth postoperative day.

Five per cent of the ophthalmologists with whom I communicated use curare to produce akinesia of the orbicularis and extraocular muscles.

Retrobulbar Injection

A few points which were mentioned in regard to the retrobulbar injection

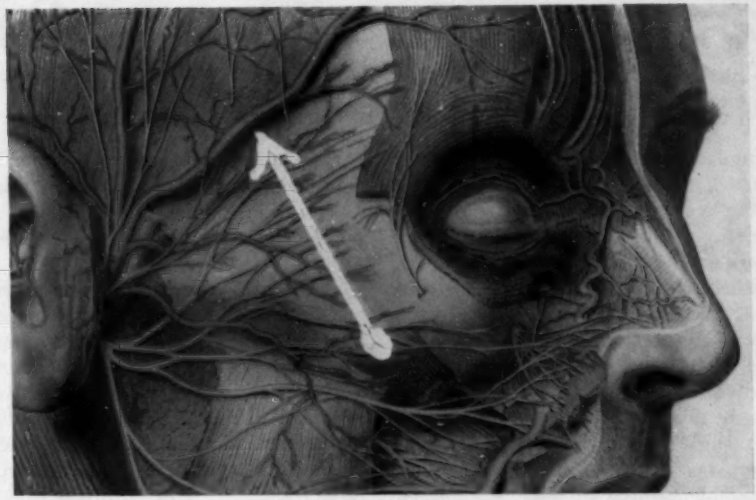

FIG. 1—Arrow shows site of injection close to zygomatic bone to produce akinesia of orbicularis muscle.

are of particular interest: the site of injection; the anesthetic solution used; the amount injected; and the use of pressure following the injection.

It is usually preferred to start the injection at the inferior temporal margin of the orbit. A few ophthalmologists give the injection at the superior temporal margin, and some inject both below and above.

The advantage of giving an injection above only is that without reintroducing the needle, the superior rectus muscle can be injected, and an injection can also be given along the superior orbital margin to produce anesthesia of the upper lid. The disadvantage is that the angle of approach is not as good as that from below because of the prominence of the superior orbital margin. Enophthalmos adds to the difficulty of making the injection above.

An injection which begins at the inferior temporal margin of the orbit has a better angle of approach and produces more satisfactory anesthesia and akinesia. A second injection above, along the superior rectus muscle, is usually necessary to obtain complete akinesia of this muscle and to prevent pain if a bridle suture is introduced. Before the needle is withdrawn after injecting along the superior rectus muscle, an injection may be given along the superior orbital margin anterior to the orbital septum to produce anesthesia of the upper lid. If these injections are not given until two to three minutes after the retrobulbar injection, as suggested by Kennedy,[4] sufficient anesthesia develops so that comparatively little discomfort will be experienced when the injection is made.

The anesthetic solutions used for injection have already been mentioned. The size of the retrobulbar injection reported as being used varied from 0.5 to 8 cubic centimeters. Before the use of hyaluronidase and pressure over the globe following retrobulbar injections was introduced, 1.0 to 1.5 cubic centimeters was the amount usually advocated. With the use of hyaluronidase and firm pressure following the retrobulbar injection, much larger injections may be

safely given. The anesthetic solution may be injected until there is a noticeable proptosis, which quickly subsides when pressure exerted over the eye is combined with moving the eye in the orbit.

Snydacker, Deutsch and Bayard[7] reported a wide variation in the akinesia of the rectus muscles and amount of hypotony obtained following retrobulbar injections of 1.5 cubic centimeters of the anesthetic solution. They attributed this to the fact that the anesthetic solution does not reach the right spot each time. This can be overcome by the use of larger injections of the anesthetic solution with hyaluronidase followed by pressure which causes the anesthetic solution to permeate the entire orbit more thoroughly, so that complete anesthesia, akinesia, and marked hypotony are more consistently obtained.

A point often overlooked is that the anesthetic effect on both sensory and motor nerves is enhanced by pressure. This applies especially to motor nerves, which are less susceptible than sensory nerves to block by anesthetic agents unless the anesthetic is applied to the nerve with pressure.[3]

The length of time advocated by different surgeons for applying pressure varies. In some patients it requires longer than in others to obtain complete akinesia of the orbicularis and rectus muscles with marked hypotony. If this is obtained, anesthesia of the globe will also be complete. Rather than setting a time limit of five, ten, or fifteen minutes to apply pressure, it would seem better to apply it until the desired effect is produced.

Kirsch and Steinman[5] emphasize the importance of pressure to produce hypotony. They report a decrease in cases which sustained vitreous loss during cataract extractions of from four in one hundred cases when no pressure was applied to none in one hundred cases when pressure was used following retrobulbar injections of xylocaine, 2 per cent, with epinephrine and hyaluronidase.

SUMMARY

Careful preanesthetic preparation and medication cannot be overemphasized.

Tetracaine hydrochloride appears to be the most popular topical anesthetic agent.

Procaine hydrochloride (novocaine) and lidocaine hydrochloride (xylocaine) are the anesthetic agents most frequently used for injection.

Epinephrine hydrochloride is usually added to the anesthetic solution unless contraindicated. Hyaluronidase is also added to the anesthetic solution by three fourths of those surgeons who use local anesthesia. The use of hyaluronidase and firm pressure over the site of injection produces more complete anesthesia and akinesia. Pressure following retrobulbar injections also increases hypotony, which decreases the percentage of cases with vitreous loss and makes the operation of cataract extraction and other intraocular operations safer and easier.

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INTRAOCULAR SURGERY UNDER GENERAL ANESTHESIA
A Study of the Problems from 1950 to 1954

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WHEN general anesthesia is employed for intraocular surgery, the patient is freed from the responsibility of maintaining his eye in any particular position, and he is no longer subjected to the stress of the knowledge that his eye is being manipulated. From the subjective viewpoint of most patients, it is the ideal form of anesthesia. However, in exchange for these great advantages to the patient, certain disadvantages have accrued to the anesthesiologist and the surgeon. Ultimately, of course, these disadvantages may work to the detriment of the patient's well-being. The time has seemed propitious, therefore, to review our experiences in cataract extraction and other types of intraocular surgical procedures performed under general anesthesia during the period from 1950 to 1954, and to comment upon both the advantages and disadvantages of this anesthetic technique.

ANESTHESIA FOR INTRAOCULAR SURGERY

The types of anesthesia which may be employed for intraocular surgical procedures are summarized in table I. Topical anesthesia, and combined topical and regional anesthesia are receiving lesser degrees of patient acceptance as the layman's knowledge of the pleasantness of pentothal induction becomes more widespread. Furthermore, some patients are simply either too nervous or too sensitive to be suitable candidates for a local anesthetic procedure. On the other hand, preoperative sedation, which when adequate is the key to successful local anesthesia, may be too massive in certain elderly patients, introducing complications of an opposite nature.

TABLE I

TYPES OF ANESTHESIA FOR INTRAOCULAR SURGERY

1. Topical (pontocaine or cocaine)
2. Combined Topical (pontocaine or cocaine) and Regional (procaine)
   a. Van Lint or O'Brien
   b. Retrobulbar
   c. Subconjunctival
3. Combined Topical (pontocaine or cocaine), Regional (procaine) and General Anesthesia (pentothal or demerol)
4. Full General Anesthesia (endotracheal)

At the other end of the scale is deep general anesthesia, produced by a variety of agents, which must, of course, be administered by the endotracheal route. While such an anesthetic technique meets with wholehearted patient approval, including as it does complete

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unconsciousness, surgical enthusiasm for the method is tempered by the occasional patient who may cough during extubation and endanger his eye by so doing. The incidence of nausea and vomiting may be slightly higher with this method than with regional anesthesia, but drugs recently made available reduce the hazard of vomiting, with its secondary effect on the eye.

A technique, therefore, has been evolved which lies between these two extremes, and which combines the advantages of comfort for the patient that is offered by pentothal hypnosis, at the same time maintaining many of the features of safety that are inherent in the techniques of topical and regional anesthesia. The method is essentially a combination of topical and regional anesthesia, with supplementary pentothal hypnosis. The term general anesthesia has been applied to this technique, since the patient is in a controlled sleep, or unconscious state, in contradistinction to the less controllable state that may obtain when preoperative sedation alone is employed, unsupplemented by further intravenous medication. Since pentothal is a hypnotic rather than an anesthetic agent, no attempt is made to conduct surgical procedures under that agent alone, without the use of topical and/or regional anesthesia to provide basic analgesia and relaxation. Furthermore, it must be emphasized that this is not a technique to be employed merely as an emergency measure because the patient cannot be managed satisfactorily under local anesthesia: both the surgeon and the anesthesiologist must have time to survey the problem well beforehand, to formulate their judgments, and to plan their courses of actions. Only under such circumstances can the best results be obtained and many complications be avoided.

THE ANESTHETIC TECHNIQUE

Patients were premedicated according to age and general physical status. The most commonly employed drugs were a barbiturate administered orally two hours prior to operation, followed by demerol or morphine and atropine injected subcutaneously one hour prior to operation. Older patients received chloral hydrate rather than a barbiturate, and the opiate was omitted preoperatively. Administration of atropine was found desirable to prevent production of excessive secretions, which stimulate coughing and endanger the surgical result. The basic pathologic process in certain intraocular conditions, such as glaucoma, contraindicate the use of atropine. In this regard, consideration must be given to the eye not to be operated on, if it is glaucomatous.

The most important factor in the prevention of complications during this technique of general anesthesia is its use in conjunction with an entirely adequate topical and regional anesthesia. This is essential in order to reduce the amount of general anesthetic agent that is required, and to obtund noxious reflexes arising in the operative area. Cocaine drops were therefore instilled in the eye several times at five-minute intervals over a period of one half hour prior to operation to produce an effective topical anesthesia. Since this serves to anesthetize the nasolacrimal duct and rhinopharynx, it may prevent coughing, sneezing, or laryngospasm caused by the irritating effects of solutions traversing this route from the eye during operation. Regional anesthesia was then combined with the topical anesthesia, a cubic centimeter or more of procaine being injected in the conjunctiva and under the superior rectus. Retrobulbar injection may be necessary, also, but if it is at all possible, this maneuver should be avoided because of the danger of retrobulbar hemorrhage.

In this series, general anesthesia was added to the basic combination of topical and regional anesthesia by the administration of pentothal by the intravenous route. The original injection generally consisted of a few cubic centimeters of pentothal in a concentration of 2.5 per cent, followed by the continuous infusion of pentothal in a concentration of 0.2 per cent. Additional supplements of pentothal in the stronger concentration were added as needed and again a few seconds before the eyeball was entered, since adequate control of the patient is essential at that time. When premedication appeared inadequate and the patient required excessive doses of pentothal, or in robust individuals who were expected to utilize unduly large amounts of pentothal, the intravenous administration of demerol was a useful adjunct.

RESULTS

Between six hundred and nine hundred ophthalmologic procedures are performed annually at the Hartford Hospital, of which approximately 50 per cent are intraocular operations. One hundred unselected intraocular procedures (table II) have been reviewed, in which the described technique of general anesthesia supplementing a combination of topical and regional infiltration was employed. Patients between sixty and seventy years of age constituted 78 per cent of the series.*

The type of cataract extraction attempted in all cases, except those of hypermature or infantile congenital cataracts, was intracapsular. If the capsule ruptured, all capsular fragments that could be removed were obtained. Forceps, rather than the erisophake, were preferred for this purpose. Iridectomy was performed more often than iridotomy. Three corneoscleral catgut sutures in the conjunctival flap were employed routinely.

TABLE II

TYPE OF SURGERY

OPERATION	NO. OF OPERATIONS
Extraction of Cataract	55
Congenital Cataract, Linear Extraction, or Discission	11
Single Operation for Cataract and Glaucoma	10
Retinopexy for Detachment	9
For Glaucoma	15
Total	100

For glaucoma, the most frequent filtering operation was iridencleisis, often in conjunction with sclerectomy. The trephine was used but once in this series. The most important complication occurring in this series was a loss of vitreous in three patients, due to coughing, straining or laryngospasm. However, no eyes were lost and the visual results were all good: 20/20 in one patient, and 20/50 in the other two.

SURGICAL CONSIDERATIONS

The technique of general anesthesia combined with topical and regional procedures which has been described often provides ideal anesthesia not only from the patient's point of view but also from the ophthalmologist's viewpoint. Under ideal circumstances, the patient is unconscious and the eye is relaxed. The patient enjoys the advantages of general anesthesia without risking the disadvantages of the nausea in the postoperative period which may follow a full general anesthesia; occasionally, when the patient has evidenced severe nervous tension, dramamine has been employed to prevent dangerous postoperative nausea and vomiting.

*Nervous tension, which is difficult to measure and record, was noted to be excessive in 18 per cent of the patients in this series.

Another advantage to the ophthalmic surgeon of this technique of general anesthesia is the complete control possible over the patient throughout the operation, irrespective of its duration. Extensive operative procedures can be performed with relative ease. Several of the patients in this series had a single operation for both cataract and glaucoma; other patients were subjected to sclerectomy combined with iridencleisis for severe glaucoma; and still other patients underwent scleral resection and retinopexy, with the separation of several rectus muscles, including an oblique. Such extensive procedures are easier for both the patient and the surgeon when general anesthesia is employed.

A final advantage to the surgeon is the lowered intraocular pressure which often accompanies the use of general anesthesia for intraocular surgical procedures. Hyaluronidase was not required for retrobulbar injection. The eyes were generally soft when the operation was begun, and even glaucomatous eyes were softer than they could be expected to be under local anesthesia. Indeed, the reduction of intraocular pressure probably equalled that obtained by prolonged manual pressure, as employed by Chandler, Dunphy, and others: that method of reducing intraocular pressure is important for operations under local anesthesia, but it is unnecessary with general anesthesia.

It is essential to emphasize that the utmost cooperation between the anesthesiologist and the ophthalmic surgeon is mandatory for the successful conduct of this anesthetic technique, for problems arise both in the realm of the anesthesiologist and in the realm of the ophthalmic surgeon which may endanger the outcome for patients, particularly among those over 60 years of age and likely to be suffering from cardiovascular and cardiorespiratory diseases. Only when both physicians appreciate and respect the other's problems and aims can this technique be employed with safety for the patient, for the technique of itself introduces inherent risks for the patient's life as well as the patient's vision. These include partial loss of airway, coughing, or sneezing before the eye is opened, straining during operation, and prolonged unconsciousness postoperatively.

In the event of severe and uncontrollable coughing during the anesthetic induction, the patient is allowed to awaken and the operation is continued under local anesthesia. Such coughing and/or laryngospasm early in the anesthetic induction may be due to fluid dropping into the pharynx during the aseptic preparation of the field. Great care must therefore be taken in this maneuver. Similarly, sneezing may be due to reflex stimulation around the eye (even strong light may cause it) and can be controlled by the proper application of local anesthesia.

The important complications are those entailing hazard to the eye during operation: the open eyeball can be lost if the patient strains, struggles, or coughs, causing loss of vitreous. During operations performed under local anesthesia alone, loss of vitreous can occur. The addition of general anesthesia does not seem to aggravate the possibility when proper safeguards are used in the surgical technique. Kirby,[3] in speaking of the loss of vitreous in cataract surgery, blames it on nervous tension in the surgeon, or on unguarded or uncontrollable movement by the patient, which ruptures the hyaloid. He states that there is no "laudable loss of vitreous," and that it is to be avoided if possible. He states that it is most often caused by increased intraocular pressure, due to muscle spasm, retrobulbar injection, increased intraorbital pres-

sure, and increased uveal pressure, among other things. He feels that spasm of voluntary muscle is a potent cause of loss of vitreous, and he used a period of preoperative training for his patients who were to be operated upon under local anesthesia. He believes that the patient should be instructed to take a deep breath, rather than admonished not to hold the breath. He also mentions that the use of epinephrine may, in some instances, add to the nervous tension of the patient under local anesthesia. One of his most pertinent comments is that the relief of abnormal muscle tone due to fear or pain can reduce the incidence of loss of vitreous. Macular edema[4] has been thought to occur after such loss or prolapse of vitreous[2] into the anterior chamber. Bellows[1] states that loss of vitreous is a frequent complication of intracapsular extraction; yet, some surgeons may be able to complete a large series of cases without vitreous loss. Townes,[5] in speaking of complications of cataract surgery, reviewed 565 operations for cataract under local anesthesia, which were performed over a five-year period, in which there was vitreous loss in 37, or approximately 7 per cent.

In the present series under general anesthesia, loss of vitreous occurred in 3 per cent of the operations. This is a favorable figure, but it requires modifications in surgical technique to permit control of the eye in the event of exigencies. When one is operating under general anesthesia, the importance of a single suture with which one can close the eyeball promptly by merely pulling one end, should be emphasized. This safeguard has been utilized twice in the present series.

Finally, the third problem of prolonged unconsciousness after surgery, due to the requirement for excessive doses of pentothal, must be considered. It is obviously of great importance from the point of view of the general welfare of patients, but there have been no ocular difficulties in this series because of an excessively long recovery period.

ANESTHETIC CONSIDERATIONS

The selection of patients to be anesthetized by this technique of general anesthesia for intraocular surgical procedures constitutes one of the major problems of the method for the anesthesiologist. The absolute contraindications to general anesthesia that are established criteria for any elective operation apply with equal force to ophthalmologic operations: cardiac decompensation, severe impairment of renal function, severe hepatic dysfunction or hepatitis, uncontrolled diabetes, marked anemia, and polycythemia, all fall in this category. These complicating factors were present in a large number of the patients in this present series (table III), as might be expected in the age groups represented: the average age was 66 years. In many instances of heart disease, anemia, diabetes, or polycythemia it is possible to ameliorate the disease by corrective therapy to such an extent that operation may be performed with relative safety at a later date.

Diseases which might be expected to increase the possibility of coughing during anesthesia, or to prevent the maintenance of a clear and patent airway, are considered relative contraindications to the use of this type of general anesthesia for intraocular procedures. These include bronchiectasis, asthma, bronchitis, pulmonary tuberculosis, upper respiratory infections, and any tumor tending to obstruct the pharynx, larynx, or trachea. If a patient coughs spasmodically during anesthesia but prior to entry of the eyeball, the anesthesia has been discontinued, the patient allowed to awaken, and the operation either postponed or continued under local anesthesia.

TABLE III

ORGANIC DISEASE PRESENT PRIOR TO OPERATION

DISEASE	NUMBER OF CASES
Hypertension (systolic pressure above 150 mm. Hg)	33
(systolic pressure above 180 mm. Hg)	14
Arteriosclerotic heart disease	15
Rheumatic heart disease	1
Cardiac decompensation	1
Diabetes mellitus	10
Emphysema	4
Asthma	3
Portal cirrhosis	1
Impaired renal function	3
Anemia	1
Severe senile confusion	1
Polycythemia	2
Diabetes associated with stress	3

There are other instances wherein the contraindications to general anesthesia are relative: the possible complications of general anesthesia must be balanced against the potential benefits to be obtained. Thus, in certain patients who have suffered moderately severe cardiac, renal or hepatic damage which cannot be corrected by treatment, general anesthesia has been undertaken on the basis of a calculated risk. Also to be included in this category are senility, extreme apprehension and psychosis in which cooperation is impossible. One contraindication of prime importance in the selection of patients for this anesthetic technique is marked obesity, particularly when it occurs in the short, thick-necked individual. Experience has shown that these patients are prone to be serious anesthetic problems because of their tendency to cough and/or develop partial or complete respiratory obstruction. In these instances it is wise to plan on general anesthesia with an endotracheal tube inserted.

In order to ascertain all the information necessary for an intelligent selection of patients for general anesthesia, it is obligatory to obtain a thorough preoperative study. This should include a complete history and physical examination, as well as basic laboratory data: complete urinalysis, blood counts and hematocrit, determination of blood sugar and of nonprotein nitrogen. Additional studies, including chest roentgenogram, electrocardiographic tracings, determination of blood volume, are obtained whenever warranted for preoperative evaluation.

The average induction and operation lasted fifty minutes, during which 1036 mg. of sodium pentothal were administered. Twenty-five patients also received from one to four cubic centimeters of d-tubocurare, and fifteen received supplements of intravenous demerol. The average patient was conscious and talking in the operating room or in the recovery room within forty minutes, the longest recorded recovery time being two hours. The average stay in the hospital after operation was nine days.

Anesthetic complications were encountered in 7 of the 100 cases.

1. Mr. D., an obese man with diabetes, received 1750 mg. of sodium pentothal supplemented by nitrous oxide and curare. He began to awaken when the needle was inadvertently displaced from the vein; it was reinserted with no ill effects.

2. Mr. H. began to cough during induction. The general anesthetic was stopped, and the operation was satisfactorily performed under local anesthesia.

3. Mr. T. received 1450 mg. of sodium pentothal. It was so difficult to maintain deep anesthesia with a good airway that it was necessary to perform an emergency intubation after administration of anectine. Anesthesia was then maintained with cyclopropane.

4. Mrs. T., a 64-year-old patient with hypertensive arteriosclerotic cardiovascular disease and blood pressure of 220/110, suffered a decrease of systolic pressure to 80 mm. Hg. Operation was postponed. At a second operation her blood pressure fell 60 mm. Hg. No ill effects were noted.

5. Mr. F., a 79-year-old patient, underwent two short periods of Cheyne-Stokes respiration (unknown etiology) with no ill effect.

6. Miss McN., a 78-year-old obese woman with arteriosclerotic heart disease and signs of mild decompensation, such as dyspnea on effort and bilateral pitting ankle edema, had stertorous breathing throughout the operation and, in spite of administration of 2650 mg. of sodium pentothal, vitreous was extruded.

7. Mr. A., an emphysematous 85-year-old male, moved his eyeball abruptly during anesthesia, and vitreous was extruded.

Miss McN. is an example of the type of patient who should not receive general anesthesia. Unfortunately, due to the patient's refusal to cooperate with local anesthesia, general anesthesia was attempted, in spite of a preoperative note by the anesthesiologist that local anesthesia should be utilized. This problem is one which must be decided on the merits of each case, and certainly every attempt should be made to attain the cooperation of such patients. Full general anesthesia with insertion of an endotracheal tube might well have been a better choice of method. By comparison, Mr. W. (not listed) was an example of a patient considered unfit for general anesthesia; yet he was operated upon twice under local anesthesia, supplemented on one occasion by administration of demerol intravenously and on another occasion by nembutal intravenously. He was 46 years old, had diabetes, weighed 244 pounds, and had arteriosclerotic, hypertensive cardiovascular disease and a blood pressure of 290/160. If this patient had refused local anesthesia, a trial of general anesthesia would have been extremely hazardous.

SUMMARY

1. An anesthetic technique has been described which employs general anesthesia (pentothal hypnosis) in combination with topical and regional anesthesia for the performance of intraocular surgical procedures.

2. The results in one hundred intraocular procedures are recorded.

3. Patient acceptance of the technique was excellent; certain problems inherent in the technique were posed for the anesthesiologist and the ophthalmic surgeon.

4. Complications, from the surgical point of view, were few, the most serious being loss of vitreous in 3 per cent of the cases. A technique was developed surgically to permit rapid closure of the eye in the event that coughing or straining occurred during the procedure.

5. Complications, from the anesthesiologic point of view, consisted of laryngospasm, coughing, obstruction to the airway, hypoxia, and hypotension. A meticulous selection of patients for the use of the technique is essential.

6. Surgeons and anesthesiologists must be prepared for a rapid change of plan: either to the use of full general anesthesia through an endotracheal tube or to local anesthesia.

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CONTINUOUS RETROBULBAR ANESTHESIA

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THIS paper presents a method for maintaining continuous retrobulbar anesthesia by repeated injections of local anesthetic solution through fine polyethylene tubing, one end of which rests in the muscle cone. The tubing is inserted into the muscle cone through a retrobulbar needle which is still in position after the injection of anesthetic solution for retrobulbar anesthesia. The tubing is held in place while the needle is withdrawn over it. Repeated injections can be made through the tubing for as long as retrobulbar anesthesia is desired.

The method was developed as a result of interest stimulated by previous work with long-lasting local anesthetic agents[11] directed toward obtaining local anesthesia of sufficient duration to avoid postoperative pain. Advantages of local anesthesia which could eliminate postoperative pain would be numerous. Pain following ocular surgery may be sufficiently severe to cause marked apprehension and restlessness, with squeezing of the lids, threshing about in bed, nausea and vomiting. Postoperative sedatives and analgesics, particularly in older people, may lead to further vomiting, a confused mental state, hypotension and other complications. Discomfort following ocular operations is usually most severe from the time the local anesthesia wears off, two to three hours after injection, until ten to twelve hours after injection. Following this initial postoperative period during which pain is frequently throbbing and intense, little discomfort occurs in the absence of complications. Local anesthesia that could be prolonged for this critical period of twelve hours, or slightly more, would be very advantageous. It would also be very helpful in other ocular conditions accompanied by protracted pain or in which repeated painful manipulations, such as paracentesis, might be required.

Although the use of retained needles or catheters, for repeated or continuous injections of local anesthetic solution to prolong local anesthesia, is rather commonplace in other fields of surgery, no reports of its employment are found in ophthalmologic literature. Lemmon,[9] 1941, apparently first used a retained flexible intraspinal needle for continuous spinal anesthesia, whereas Edwards and Hingson,[6] 1942, used an identical method for continuous caudal anesthesia. Manalan,[10] 1942, and Adams, Lundy and Seldon,[1] 1943, unaware of each other's work, independently introduced the use of a retained catheter inserted through a needle for continuous caudal anesthesia. Touhy,[14] in 1945, first used a catheter for continuous spinal anesthesia. In 1949, Thomason and Moretz[13] used the same technique for continuous lumbar paravertebral sympathetic block. Blades and Ford,[4] 1950, introduced the method for thoracic surgery. They placed polyethylene tubing in the wound at the site of painful ribs for control of postoperative pain in chest surgery. Procaine solution injected through the tube every two to three hours gave considerable relief from postoperative pain and

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discomfort. The tubes were left in place from three to nine days. No infection or interference with wound healing was noted in 36 patients, 18 of whom served as controls. Gerwig, Thompson and Blades,[7] in 1951, employed the same technique following upper abdominal surgery, feeling that reduced pain would ensure freer breathing and fewer respiratory complications. Catheters were placed beneath the anterior rectus muscle sheath of abdominal wounds and brought out away from the wounds through stab incisions. Three cubic centimeters of 2 per cent procaine solution was injected through the tubing at three-hour intervals. Gerwig and his co-workers studied its effectiveness on 24 patients who had had cholecystectomies, 12 of whom served as controls. The pain ordinarily was greatest between the twelfth and thirty-sixth postoperative hours and was largely gone by the eighty-fourth hour. The control group needed eleven hypodermic injections per person, whereas the treated group required only three injections during the period of acute pain. The tubes were removed at the end of sixty hours with no ill effects being noted. Gerwig, Alpert, Coakley and Blades,[8] in 1953, further extended the use of continuous caudal anesthesia maintained by polyethylene tubing for relief of pain following hemorrhoidectomy. Anesthesia was continued for forty-eight hours with striking relief from pain. No complications were encountered in 59 patients. Ansbro and his co-workers,[2] in 1952, used continuous spinal anesthesia for a period of eleven days with no ill effect in the case of a patient with a vascular lesion of one lower extremity. Sergent and Owen,[12] 1951, mentioned its use for serial stellate ganglion block. Davidson, Hingson and Hellman,[5] in 1951, reported having used plastic catheters in the subarachnoid and peridural spaces of 1,913 patients for the continuous administration of anesthetics and therapeutic medicaments to control pain from such a variety of conditions as postdelivery, advanced malignancy of the prostate, carcinoma of the uterus, severe rectal fistulas, severe vulvovaginitis following radium burn, and acute pancreatitis. The catheters were left in place from a few hours to 142 days. One patient developed cellulitis over the sacrum, and one developed aseptic meningitis following introduction of a polyethylene catheter. Data gained from the literature indicate that continuous local anesthesia by the use of a retained needle or catheter is safe and very practical. It is now extensively used for prolonged spinal and caudal anesthesia, and the technique is widely accepted.

TECHNIQUE FOR CONTINUOUS RETROBULBAR ANESTHESIA

Local anesthetic solution is injected through a 27-gauge needle to raise a wheal in the skin over the lateral inferior aspect of the lower lid, and the lid septum is infiltrated to prevent pain when passing the retrobulbar needle. A specially made, thin-walled 19-gauge needle, containing a stilet to ensure patency, is used for retrobulbar anesthesia (figs. 1 and 2). The finest polyethylene tubing available, PE-10, readily threads through such a needle. The smallest standard needle of regular thickness through which the tubing will pass is 18-gauge, but because of its caliber, it causes more trauma and is difficult to insert. The tip of the needle is bevelled and rounded as suggested by Atkinson.[3] After insertion of the needle into the muscle cone, the stilet is removed, a hypodermic syringe is attached, and the retrobulbar anesthetic is given. A piece of polyethylene tubing two and one-half times the length of the needle is pushed into the lumen of the needle until the resistance of orbital tissue is encountered, indicating that the

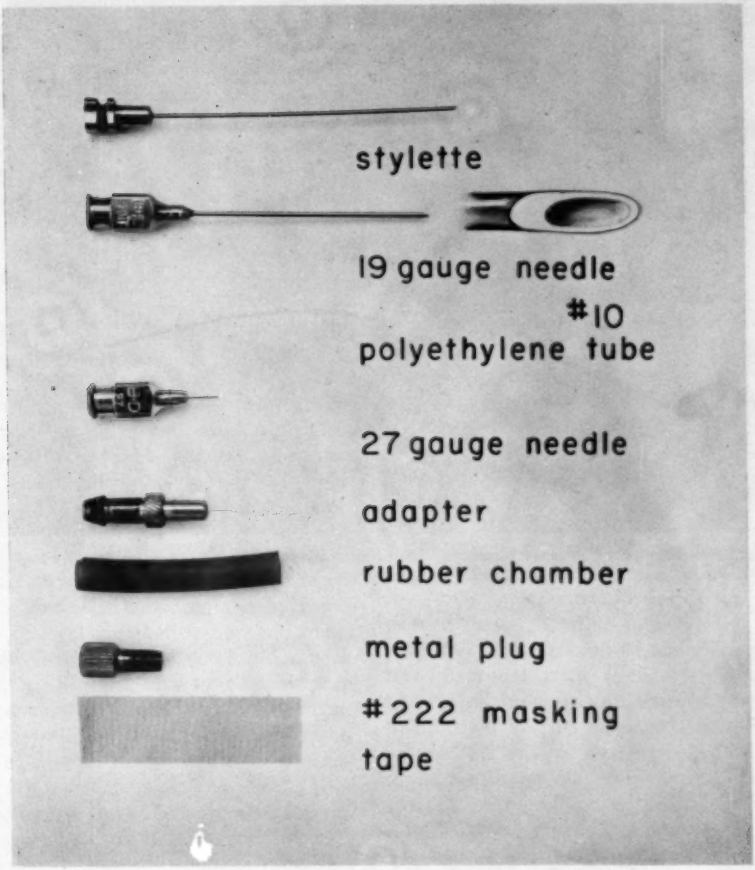

FIG. 1—Apparatus for continuous retrobulbar anesthesia unassembled.

end of the needle has been reached. In order to maintain the tubing in this position while the needle is withdrawn, the thumb and forefinger gently grasp the tube near the base of the needle as the needle passes over it (fig. 3). After the needle has been removed, a chamber containing anesthetic solution is connected to the free end of the tubing by a 27-gauge needle which fits tightly enough to form a water-tight connection (fig. 3). The chamber is made from a piece of rubber tubing, 35 mm. long, of the type and size used for intravenous injections (fig. 2). The chamber is formed by closing one end of the tubing by a metal plug and the other by an adapter that fits into the 27-gauge needle. The chamber is filled with anesthetic solution to displace all air and is sutured to the skin in front of the tragus of the ear, a convenient location because

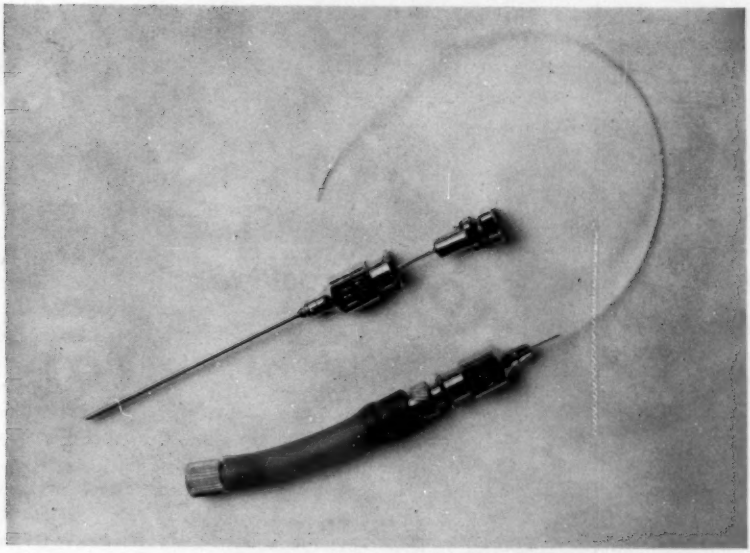

FIG. 2—Thin-walled 19-gauge retrobulbar needle with stilet and anesthesia chamber assembled.

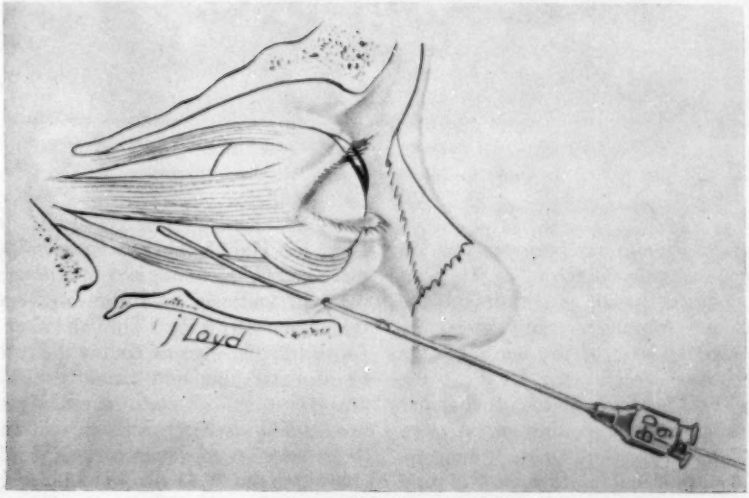

FIG. 3—Retrobulbar needle withdrawn over polyethylene tubing in muscle cone.

the skin already has been anesthetized by the O'Brien block (fig. 3). Injections of anesthetic solution can be made into the chamber by inserting a fine needle through the rubber wall, just as one injects solutions into an intravenous tubing. The wall of the rubber chamber is first wiped with an alcohol sponge. Injections can be made through the free end of the polyethylene tubing rather than employing a chamber, but a closed system probably helps to safeguard against infection. If the tubing is injected directly, the end should be sponged with alcohol, and one-quarter inch removed with a sterile scissors to ensure sterility. In either instance, the tubing is secured to the cheek by a strip of pressure-sensitive Scotch tape, no. 222, which can be autoclaved (fig. 4).

CLINICAL OBSERVATIONS

The author has used continuous retrobulbar anesthesia, maintained by injections of local anesthetic solution through polyethylene tubing placed in the muscle cone, in 125 patients to control postoperative pain. Discomfort almost invariably is relieved seconds after the injection of 2 cc. of the local anesthetic agent and this relief persists for the duration of action of the anesthetic agent. After the second or third injection, relief of pain usually lasts for a shorter time. Preoperative medication in this series of patients consisted of sodium pentobarbital, 0.6 grams, given two hours and one hour preoperatively, and dramamine orally, 100 mg. every four hours for six doses, beginning four hours preoperatively. No sedatives or analgesics were used postoperatively. Anesthetic solution was injected only if the patients complained of pain, whereas ordinarily we would have prescribed acetylsalicylic acid or meperidine hydrochloride.

Thirteen per cent of the patients had no postoperative pain and required no injections of local anesthetic solution. The remainder, or 87 per cent of patients, complained of pain and were given one or more injections. Twenty-nine per cent required only one injection. Another 23 per cent required a second injection; and 27 per cent, a third injection. Only 6 per cent required four or more injections. The average length of time following retrobulbar anesthesia until they complained of pain was two hours and forty minutes. The period of most severe pain occurred during the interval between two and twelve hours after anesthetization. Few patients had severe pain after that time. A small number of patients were given repeated injections at hourly intervals following operation with reduction or prevention of postoperative pain.

Continuous retrobulbar anesthesia was employed also in certain other conditions involving protracted ocular pain. It proved to be of value in relieving the pain and apprehension of acute congestive glaucoma until medical treatment became effective or until surgical treatment could be carried out. It also embodies the tension-lowering effect of a local anesthetic and epinephrine. Continuous retrobulbar anesthesia is also helpful when repeated minor operative procedures are necessary over a period of days. An excellent example of its usefulness occurred in the case of an eye with traumatic hyphemia and secondary glaucoma for which repeated paracenteses were necessary. The patient was a nervous youngster, fifteen years of age, who would not have cooperated in repeated retrobulbar anesthesia and manipulations of his eye. Furthermore, he had recurrent severe pain at times during the intervals between the operations on his anterior chamber. The tubing was allowed to remain in place in his muscle cone for one week, during which time instillations were made as indicated.

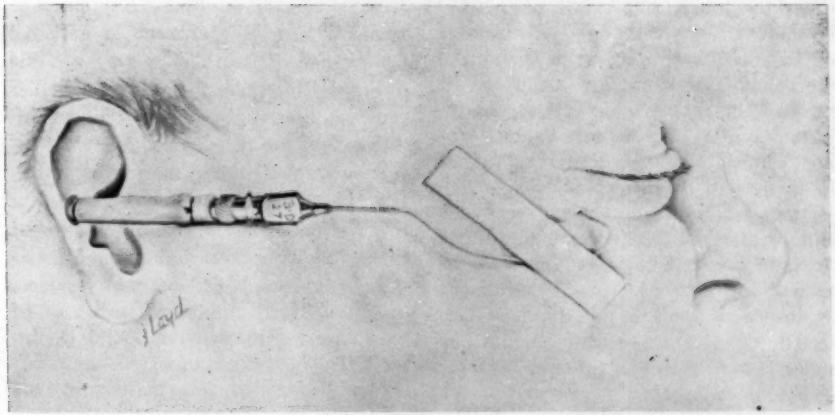

FIG. 4—Anesthesia chamber sutured to skin anterior to ear and connected to polyethylene tubing. Tubing attached to malar region by pressure-sensitive no. 222 Scotch tape.

This permitted paracentesis to be done painlessly on two occasions and relieved severe pain at other times.

Possible infection from a retained catheter must be considered, but it did not occur in our series of over 125 patients, and it is almost unmentioned in reports of continuous anesthesia in other fields of surgery. We have employed the method only for patients receiving wide-spectrum antibiotics systemically, a precaution we take after all intraocular surgery. This undoubtedly adds to the safety of the method. The method is certainly contraindicated if ocular infection is present, particularly if it is suspected that the infection is due to an antibiotic-resistant organism. Sterility must be carefully observed in inserting the tubing and in making the injections. Hemorrhage should occur no more frequently than with ordinary retrobulbar anesthesia because the tubing is pliable and hardly capable of causing damage to the blood vessels. Polyethylene tubing is nonirritating, at least for the duration of time employed in continuous anesthesia. In the vast majority of patients, the tubing has been left in place for only twenty-four hours. We have left the tubing in place for several days to a week in only a few patients, but no complications resulted. General surgeons do not hesitate to continue it for several days to a week. There have been no complications observed in the entire series. We left the tubing in place for two or three days following enucleations, depending upon the severity of the discomfort.

Continuous retrobulbar anesthesia is sufficiently simple to be of practical value. The technique is easy to learn and the equipment is readily available. It gives prompt and usually complete relief from postoperative pain for the duration of action of the anesthetic. It offers an excellent method for the study of ocular pain, including the duration of action and effectiveness of local anesthetic agents. The method can be helpful in any situation in which pain is prolonged or in which repeated manipulations of the eye are needed.

CONCLUSIONS

Continuous or repeated retrobulbar anesthesia, by injections through fine

polyethylene tubing inserted into the muscle cone, is practical and safe. The method can be helpful in any situation in which pain is prolonged or in which repeated manipulations of the eye are needed.

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DISCUSSION OF PAPERS ON OCULAR ANESTHESIA

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BY INVITATION

I THINK that the next few years will really confuse most of us because the chemists, especially in Europe, are busy on a galaxy of compounds "all of which are better than what you are using today." I have many drugs that are better than procaine. There is going to be confusion. I think that Dr. Swan is absolutely correct. Many of these drugs have distinct advantages over cocaine, over procaine, over any of the drugs that you are using today. And it is interesting that the drugs which he mentioned are also now, according to some pharmacologists, passé.

It seems to me, however, that the average practicing clinician must hold off trying this, that, and the other until large bodies of data—not 100 cases, not 500 cases, not even 1,000 cases, but truly thousands of cases—have accrued. In order to evaluate a drug or technique, it must be tried under various circumstances in many clinics before the true picture begins to be clear, and this occurs only after years of usage.

I know that Dr. Daniel Moore, in his new book Complications of Regional Anesthesia, will comment on one disadvantage that we have found with cyclaine. It lasts much longer in tissues than procaine, but every once in a while it has a slightly deleterious effect in the skin itself. So if you are using cyclaine, especially in the 2 per cent solution with epinephrine added, in spite of the fact that it "takes" a little more rapidly, in spite of the fact that it is certainly a nontoxic drug from the standpoint of local reaction, I would recommend that you go easy on skin infiltration in the debilitated aged individual. Use 1 per cent without epinephrine in the skin itself.

As far as xylocaine is concerned, I know that official bodies here in America are beginning to be concerned about the use of xylocaine in a higher concentration than is necessary for the work to be done. Do not use 2 per cent unless you need it. Remember that 2 per cent probably has no place except in nerve trunks larger than my finger, such as the sciatic nerve. One per cent is quite satisfactory for most of the operations around the eye. Xylocaine acts fairly rapidly, spreads nicely, lasts adequately, and only becomes toxic in high concentrations and volumes. It is an excellent drug for eye surgery if used correctly.

The toxicity of these agents will definitely diminish as the concentration is reduced. The important consideration is to use the proper agent in the correct concentration to obtain the necessary result. Don't send a man on a boy's job. Ten per cent procaine? I would consider it almost criminal to use concentrations above 5 per cent. Why? Because as the concentration is increased above 5 per cent, the neuritic phenomenon definitely starts to appear, and with a 7 per cent concentration, it is not too unusual to find evidences of degeneration in a section of nerve.

The justification for use of the higher concentrations, I don't understand.

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There are many longer-acting agents. If you want sustained anesthesia, use the agent that gives you the time you need. Do not be misled concerning the value of high concentrations; they add relatively little to duration of anesthesia and increase the danger of toxic reactions.

As far as hyaluronidase is concerned, it shortens the duration of anesthesia. It increases successful blocks, especially those more difficult to obtain. However, if epinephrine is added to the solution, the effect of hyaluronidase on the time factor will be negated. Toxicity from hyaluronidase is so rare as to be unimportant, but this drug should be used when its spreading factor is really needed.

If you want to induce motor paralysis, you must use an agent in a concentration strong enough to give motor paralysis. I have seen surprise because 0.5 per cent procaine did not result in good motor paralysis in very heavy-faced, large individuals. Remember that it takes a greater concentration to penetrate heavy, large nerve fibers than it does small fibers, and that the function of these nerve fibers is reduced in an orderly pattern: first, sympathetic fibers (involving the experiences of pain, temperature, and light touch); then the proprioceptive fibers, and last, the motor fibers. So if you want complete motor paralysis, you should use true deep anesthetic agents, or the weaker agents in higher concentrations, or any of the newer anesthetic drugs which give motor paralysis when used in usual concentrations.

I found the observations by Dr. Atkinson very interesting, and I agree with him wholeheartedly.

I want to emphasize that a few carefully chosen words are as potent as any sedation. I am sure that there are deaths occurring today, in fact, I have seen them myself, due to oversedation. Vocal is about as good as local. I am sure you know that in many instances you can carry out these techniques with hypnosis alone. In fact, in case of pain due to carcinoma, I frequently inject normal saline instead of a local anesthetic agent, because so high a percentage of patients say, "My pain is gone," when all I have given them is a little normal saline.

I have seen one death which was actually due to careless use of thorazine. Thorazine is an excellent sedative. By the way, I recommend to you also almost all of the sedative, antihistaminic agents such as phenergan, chlor-trimaton, benadryl—the whole galaxy. All of these drugs are excellent sedatives. The disadvantages are relatively small, if one is acquainted with them. The danger from thorazine is not from the drug but rather from a lack of knowledge as to what it does. Even though thorazine is used in dosages that are hardly more than slightly sedative, profound depression may result if the usual quantities of the narcotics are used with it. This may mean hypoxia; this may mean cerebral thrombosis; many undesirable things can occur. I recommend caution in the use of thorazine when combined with narcotics. Carefully add the sedatives, hypnotics or narcotics in divided doses until desired results occur. Thorazine increases the duration and depth of action of narcotics. Experience is the best guide and should be acquired slowly.

As far as the barbiturates are concerned, may I condemn the timing that is frequently used. If the barbiturates are to be used, give them hypodermically an hour, at the very least, before the time of surgery. Preferably give nothing by mouth if this can be avoided.

We should not be too surprised when nausea and vomiting occur, even though

the patient has been brought into the operating room merely for examination. After all, we have all witnessed the mother of a patient nauseated and vomiting outside the operating room; she was not being operated on. I have seen the father of a patient fall and require more sutures than the baby. These were reactions to the operations although no drugs were administered. Most of the reactions are not reactions to the drugs; they are reactions to the procedures. They are simple syncopes; they are not even local anesthetic reactions.

The local anesthetic reactions that you think you see are most frequently "fainting episodes." I believe that true reactions should not occur in more than one in 1,000 cases. If someone tells you that he is sensitive to local anesthetics, try to find out which drug was used and choose a drug of another type. Premedicate with barbiturates carefully and give the newer drug in very dilute concentrations and in divided doses slowly. In the case of a very sick patient, it might be better to use general rather than regional anesthesia, especially if there have been several "reactions to local."

I know of no adequate test for local anesthetic sensitivity. If an intradermal wheal is raised in testing reaction to a drug, by all means don't inject that drug into tissue. In other words, if drug X injected into the skin produces a big flare and a wheal, don't use drug X for a retrobulbar anesthetic. It is obvious that the tissues are reacting abnormally. They are being irritated by the drug. It has no place in the anesthesia program for this individual.

However, unfortunately individuals who are sensitive to one drug are usually sensitive to many agents. The more times that a specific drug is given to an individual, the more likely he is to develop a sensitivity to it.

I would also plead that you all caution your staffs regarding tests for sensitivity. Each of you knows better, I know; but in the last two years, my attention has been called to three cases of extreme irritation of the eyes, caused by a test for sensitivity made by placing the drug in a conjunctival sac. This is a nice way to observe sensitivity, but personally I feel that it has no place in modern medicine.

As far as epinephrine is concerned, we believe that reactions of palpation and tachycardia are often due to the fact that too much epinephrine was given. The concentration of epinephrine that should be used is 1 in 100,000, and it should be accurately measured. However, on a study of a group of volunteers, it was found that if you gave an injection, but the subject did not see the needle, he showed only a slight response to the injected epinephrine; however, if you showed him the needle and you said, "I am going to stick you," the response was as great, if not greater. The danger from an epinephrine injection is not great, provided it does not enter a vessel, and it would seem to me that for a deep retrobulbar injection one must aspirate to be sure that a vessel has not been entered. For this reason, all syringes should be the type suitable for aspiration. Interesting studies have recently been made of operative procedures about the face; one author will report that in 6 per cent of the cases in which there were blocks of a mandibular nerve, the needle was intravascular. This may be the cause of reactions.

I should like to condemn rapid injection. Almost all of my staff have to be slowed down on the speed of injection. The size of the needle is not the most important thing; it is the type of point used and the gentleness with which it is used. Speedy injections are always painful. Speedy injections "take off" in

tissue, and I recommend to all of the authors, who seem to have a wonderful insight into methods of study, that they study the spread of diodrast combined with local agents. If they inject rapidly, it will track far beyond their wildest imagination. Some members of our staff have had the misfortune to cause ocular motor paralysis by too rapid injections of alcohol. If the injection is made slowly, the alcohol spreads centrifugally; if it is made rapidly, the drug tracks along planes of least resistance.

As for general anesthesia, I make one very important plea. At the Illinois Eye and Ear Infirmary we have been using general anesthesia and we will continue to use it on anybody who requires it. However, at the Harvard Medical School, which in my opinion has one of the world's outstanding departments of anesthesiology, they are saying, if I may interpret it, that the hazards in general anesthesia are much greater, and one should use it with extreme caution. The risk is directly proportional to the skill and energy of the surgical team.

No patient should be refused general anesthesia because of our prejudice against it. After all regional attempts fail, we always turn to general anesthesia. General anesthesia can be tolerated by anyone but those most seriously ill, that is, those in cardiorespiratory failure. This method of anesthesia requires the teamed efforts of the surgeon, internist and anesthesiologist, and the skill of all members must be utilized or the price paid for general anesthesia will be high.

I will not permit my residents and my staff to use pentothal, or any form of general anesthesia that resembles pentothal-nitrous anesthesia, unless an adequate retrobulbar block has first been secured. I know of two deaths from this drug in England in 1943. I have had reported to me no less than five deaths from the same cause within approximately 600 miles of Chicago. If pentothal is given without adequate topical anesthesia and without adequate retrobulbar block, there is a possibility that impulses resulting from stimulation of the conjunctiva and other tissues in the area may produce a sneeze or a cough. Such impulses may reach the vagus fibers and thus arrest the heart. It is my firm conviction that all general anesthesia should be done using adequate retrobulbar block and topical anesthesia supplement unless these are absolutely contraindicated, in which case I should prefer not to use the barbiturate techniques. Pentothal is a wonderful agent when used correctly. It is probably the ideal supplement to nitrous oxide. However, if it is used incorrectly by untrained individuals, occasionally the patient may pay the superior price.

I agree with the authors that the antinausea and the antivomiting drugs really aid in the emergence period. However, some patients will still experience nausea and vomiting; for them the technique which Dr. Scheie presents is an excellent one. We have used this technique for carcinoma patients, but not in cases related to ophthalmology. The only disadvantage that I found was that we have had difficulty in maintaining the catheter in place. I think that the problem is to prevent drift of the catheter forward and back. I don't know what technique others use for fixing the catheter. We have actually taken a stitch in the skin to hold the catheter in place. We have also taken the trouble to put antibacterial agents around it.

SUMMARY

A great deal of information is being made available on anesthesia, but in the final analysis each man uses that technique which he knows best. As a patient, I would not dictate the drug to be used. Certain drugs that are coming along cer-

tainly are more potent, more rapid acting. Some of these drugs actually act almost within seconds—deep penetrating, very little irritation. They are safer if one uses the correct total dosage and correct concentration. The importance of the hazard of intravascular injection cannot be overemphasized.

I believe that general anesthesia has a tremendous place. Although some doctors say, "Oh, I don't like a general anesthesia," when they have a psychopathic patient, a child, an individual who is uncooperative, or a bouncer, they are glad to "put him to sleep." I feel that a patient of that kind should be given a general anesthetic beforehand; do not try a regional and then abandon it after it has failed. It is better to have good general anesthesia than poor regional anesthesia.

At the Illinois Eye and Ear Infirmary, any patient who wants to "sleep" is permitted to have general anesthesia. Basically, however, let's not forget that in the long run, regional anesthesia is safer than general anesthesia. I sincerely believe that much more skill is needed to do general anesthesia for eye surgery than to do many of the other forms of anesthesia. Eye anesthesia should be absolutely under control and as light as is consistent with the work at hand.

I personally will not do eye anesthesia unless an endotracheal tube is in place. However, there are many things that I don't like about local anesthesia procedures in your specialty. I would recommend to many of you that you get up on that table, which you think is such a nice table, and just lie there for an hour. I get fidgety. I cannot even rest there more than thirty minutes without feeling extremely uncomfortable. You should provide better comfort for the patient.

Also, there are sheets and all kinds of things put over the patient's face. Sometimes he is fidgeting, not because you are hurting him but because he cannot "catch his breath." Oxygen lack and carbon dioxide excess are probably occurring in many of these patients.

It often seems to me as I watch eye surgery, that the attitude is, "If the patient does not bother you, don't you bother the patient." Sometimes I have wondered, as I have watched, whether the patient was alive, and if not, how much this was due to the extreme sedation. This sounds like a joke, but we know it is true. Avoid oversedation; it is dangerous. It may actually cause restlessness rather than prevent it, especially if hypoxia and confusion occur.

It is my hope that you will soon be giving the same consideration to your patients as is afforded the patients of other surgeons; i.e., that a blood pressure cuff be put on the patient; that pulse and blood pressure be taken; that fluids be given. I believe that many of the coronary thromboses and cerebral vascular accidents that happen three, four and five months after an eye operation really are due to the hypotension caused by excess sedation during the eye operation and to inadequate observation and support during the eye operation.

OCULAR LESIONS OF NEVOXANTHOENDOTHELIOMA (INFANTILE XANTHOMA DISSEMINATUM)

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THANNHAUSER'S extensive clinical and laboratory studies of lipoidal diseases[5] have improved our knowledge of these conditions so that isolated bits of information about apparently separate clinical entities can now be made into an organized classification of related syndromes. The following is a simplified classification of cutaneous xanthomas in children, which was suggested by Crocker[2] and which conforms to the general concepts of Thannhauser:

Type I.
Serum Cholesterol (and Fats) Normal:

Group A. Xanthoma disseminatum (including those originally called "nevoxanthoendothelioma" and juvenile xanthoma)

Group B. Xanthomas accompanying various chronic diseases, especially the "eosinophilic granuloma syndromes" or the so-called "reticuloendothelioses" (including Hand-Schüller-Christian disease and Letterer-Siwe's disease)

Type II.
Serum Cholesterol (and Fats) Elevated:

Group C. Xanthoma tuberosum (including essential familial hypercholesterolemia)

Group D. Secondary or eruptive xanthomas (including the transitory xanthomas of diabetes mellitus, idiopathic familial hyperlipemia, nephrotic syndrome, hypothyroidism, obstructive liver disease and von Gierke's disease)

Hand-Schüller-Christian disease, Letterer-Siwe's disease, and eosinophilic granulomas of bone were at one time thought to be separate disease entities, but the present trend is to consider these conditions as different phases or degrees of involvement of a disturbance of the reticuloendothelial system. The histologic appearance of the skin lesions in nevoxanthoendothelioma (disseminated xanthomas in infants) are similar to those seen in the former conditions. However, systemic lesions had not been observed prior to a report by Blank, Eglick and Beerman[1] of a case in which there was ocular involvement in the form of a granuloma of the iris and ciliary body in one eye. The histologic appearance of the skin and eye lesions in this case were almost identical. These authors predicted that other cases of nevoxanthoendothelioma in which there was systemic involvement would be found, thus integrating the syndrome more closely to other eosinophilic granulomas.

CASE REPORT*

History: This white male child (D.F.), aged 4 months, was first seen by an ophthalmologist on February 23, 1953, because of epiphora and steaminess of the right cornea. The eye had been involved for two to three weeks. Examination by a pediatrician revealed the child to be normal except for multiple xanthomas of the skin and difficulty with the right eye.

Eye Examination: Examination while the child was under ether anesthesia showed the left eye to be entirely normal. The right cornea was steamy, and there were many "fat-

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Presented as a *Clinicopathologic Case Report* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

*Dr. Carl Jensen of Seattle, Washington, was the ophthalmologist in charge of the case and kindly gave his permission to report this material.

like accumulations" on the posterior surface of the cornea and anterior surface of the lens. There was some fresh blood in the anterior chamber. The iris was thickened and dirty white in color in some areas. The intraocular pressure measured 60 mm. Hg (Schiøtz). Paracenteses were performed on February 24, February 27, and March 9. The ocular tensions remained elevated and the cornea steamy. The iris became thicker and more involved by an apparent new formation of tissue. Several fresh hemorrhages were noted in the anterior chamber.

The eye remained inflamed, and the child was irritable and obviously in pain. By May 7, 1953, it seemed impossible to save the eye because of the thickened iris and uncontrollable glaucoma. The possibility of a tumor of the eye could not be ruled out, so the eye was enucleated and a biopsy of the skin performed. The material was sent to the Stanford Unit Eye Pathology Laboratory for study.

In May 1955, word was received from the child's mother that he was healthy, the left eye was normal, and the skin lesions had begun to subside.

Report on Pathological Material

Right eye, Gross: The right eye measures approximately 23 by 24 by 23 mm. and has about 1 mm. of optic nerve attached. The cornea is slightly cloudy, and there are fragments of whitish material in the anterior chamber. Transillumination of the globe revealed no abnormalities. The globe is sectioned vertically and is filled with normal-appearing vitreous. The angle of the anterior chamber is filled with white coagulated material which forms a layer over the iris and pupil. The lens is intact. The retina, choroid and sclera in the posterior segment of the eye appear to be normal. The disc is moderately cupped.

Right eye, Microscopic: The optic nerve, retina, choroid and sclera behind the pars plana are essentially normal. There is no evidence of abnormal cupping of the optic nerve. The anterior segment of the eye contains pathological findings of interest (fig. 1). The iris, ciliary body and trabecula are almost completely replaced by dense infiltration of cells containing a large pale-staining oval nucleus with many nucleoli and slightly eosinophilic-staining cytoplasm. These cells are not arranged in any particular pattern but merely replace most of the normal cells and stroma in the areas described. Numerous newly formed vessels are noted throughout the stroma of the iris and ciliary body. A great number of Touton giant cells are found in the reaction, but they are especially noticeable on the surface of the iris and ciliary body next to the anterior chamber (figs. 2 and 3). Also on the surface of the iris, the cells appear to be breaking off into the aqueous. Fresh hemorrhage and old blood pigment are present in the angle of the anterior chamber and dissect Descemet's membrane from the posterior surface of the cornea in the central area. Newly formed connective tissue is noted between the detached Descemet's membrane and normal corneal stroma. A number of eosinophils are noted in the angle of the anterior chamber. The trabecula, Schlemm's canal and the adjacent sclera in the region of the deep scleral vessels are markedly infiltrated by histiocytes. These cells are also present in the posterior chamber covering the ciliary processes; however, the pigmented and nonpigmented epithelium of the ciliary body appears intact, and the pigment epithelium of the iris is undisturbed. No foam cells are seen in this reaction.

Skin: Biopsy of the xanthoma of the skin shows it to be covered by normal stratified squamous epithelium, the Reedy pegs of which are well delimited by the basement membrane. In areas, the dermis is almost completely replaced by histiocytes similar to those found in the eye (figs. 4 and 5). Numerous Touton giant cells are scattered throughout the reaction. No foam cells or polymorphonuclear cells are noted.

DISCUSSION

The case just reported confirms the prediction of Blank, Eglick and Beerman that other instances of systemic involvement would be found in patients with nevoxanthoendothelioma. In this case, no organic lesions were found on routine pediatric examination other than those of the skin and the eye. Special studies were not done, because the correct diagnosis was not made for almost a year after enucleation, and by this time the child had moved to another community.

Engelbreth-Holm, Teilum and Christensen[3] have noted that eosinophilic granulomas progress through the following four phases:

1. A proliferative phase, which consists of histiocytic proliferation with accumulation of eosinophilic histiocytes. Foam cells are absent.

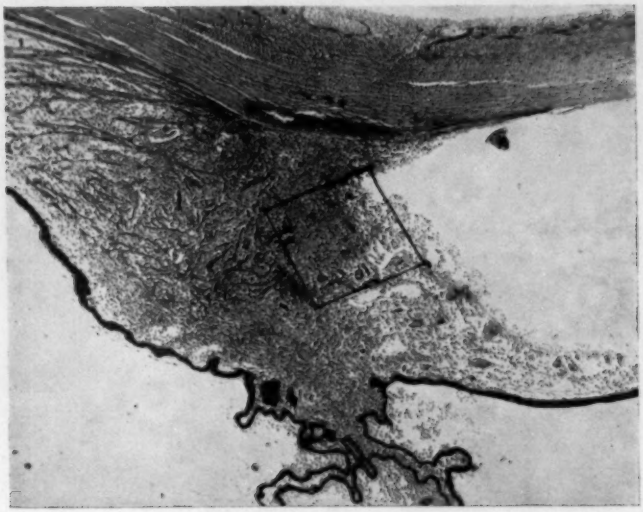

FIG. 1—Section of eye. Infiltration of iris, ciliary body and trabecula by histiocytes, Touton giant cells and eosinophils. (×40)

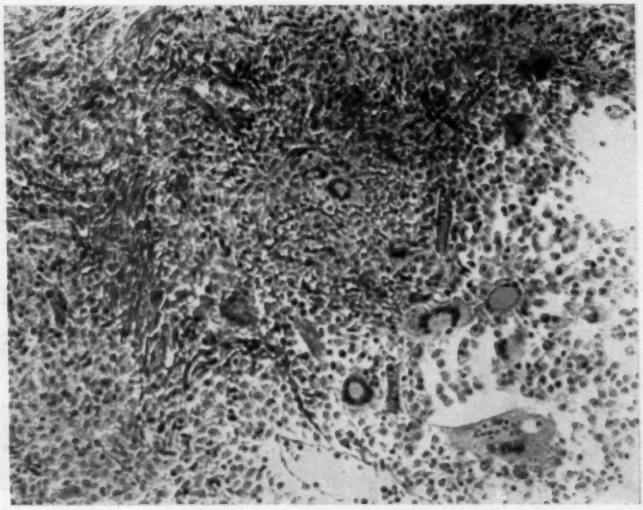

FIG. 2—Same as figure 1. (×120)

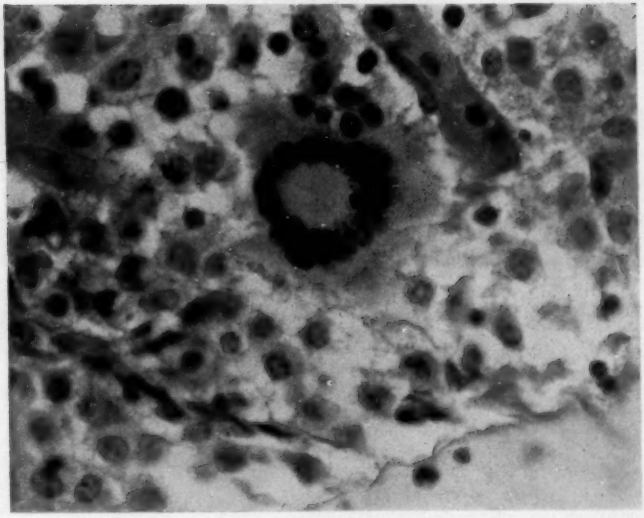

FIG. 3—Same as figure 1. (×540)

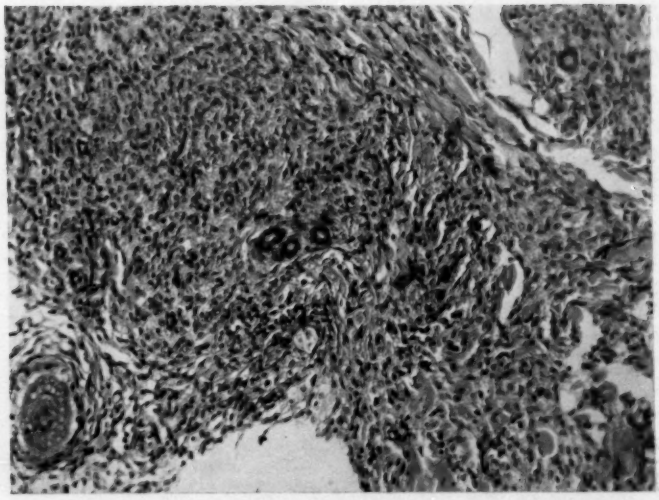

FIG. 4—Section of skin, showing dermis almost replaced by histiocytes with Touton giant cells. (×120)

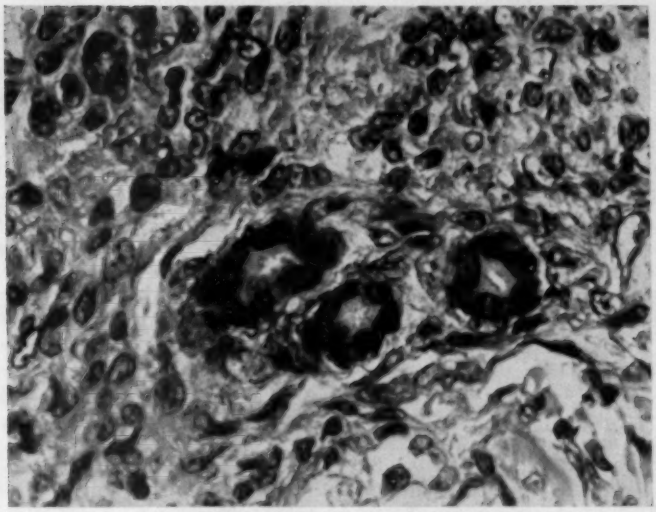

FIG. 5—Same as figure 4. (×540)

2. A granulomatous phase, in which histiocytes and Touton giant cells predominate. The blood vessels and fibrils increase and eosinophils appear. The macrophages show incipient accumulations of lipids.

3. A xanthomatous phase, accompanied by nests of foam cells.

4. A fibrous stage, or healing phase.

The lesions of the patient in the case just described were in the early granulomatous phase at the time of the skin biopsy and enucleation.

It is interesting that intraocular involvement has not been reported in patients with Hand-Schüller-Christian disease, Letterer-Siwe's disease, or eosinophilic granuloma of the bone. However, granuloma of the anterior uveal tract has been the most frequent systemic involvement in nevoxanthoendothelioma. The occurrence of this intraocular involvement is probably not as rare as it might appear from a review of the literature. Sanders[4] has recently collected a half dozen or more cases of this syndrome which he will report in the near future.

In all of the cases which have come to the attention of the author, spontaneous recurrent anterior chamber hemorrhages and yellowish white thickening of the iris have been a common finding. Secondary glaucoma usually occurs a few weeks after the eye becomes involved. Because of the rarity both of spontaneous anterior chamber hemorrhage and of marked thickening of the iris in infants, any child under the age of three with these symptoms should be carefully studied for xanthomas of the skin, and a diagnosis of nevoxanthoendotheliomas should be suspected.

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DIKTYOMA OF THE CILIARY BODY

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A DIKTYOMA is a malignant tumor of the nonpigmented epithelium of the ciliary body, having a histologic appearance which resembles that of embryonic retina prior to stratification. The tumor is uncommon, and Andersen[1] in a 1948 review found but twenty-two examples, of which the diagnosis was considered uncertain in seven. Since then, additional cases have been reported by Fralick and Wilder[4] and Malone.[7]

The primitive medullary epithelium of the neural tube, which lines the secondary optic vesicle, gives rise to the pars optica retinae, pars ciliaris retinae, and pars iridis retinae. About the second developmental month, the pars optica retinae portion begins differentiation into the various nerve layers of the retina.

The medullary epithelium of the pars ciliaris retinae persists in a relatively undifferentiated form, and tumors arising from this epithelium are similar to the medulloepitheliomas of the brain. Grinker[6] in 1931 applied this term to such tumors. Theoretically, such a malignant tumor could also arise from the iris but there have been no reports of an origin in this location. A diktyoma arises from undifferentiated medullary epithelium; a tumor arising from the epithelium after maturation is classed as a malignant epithelioma and is commonly preceded by ocular inflammation. The diktyoma was first described by Badal and LaGrange,[2] who regarded it as an adenocarcinoma. Emanuel[3] described a similar tumor in a 5-year-old boy; he considered it to be a glioma. Verhoeff[8] in 1904 was the first to appreciate the exact nature of the tumor, and he proposed the term "terato-neuroma." He described the main features of the growth:

(1) Intricate convolutions of two types:
  (a) a single layer of columnar epithelium in direct continuity with normal unpigmented epithelium.
  (b) several layers of cells, not arranged in parallel rows, resembling embryonic retina.
(2) a sharply defined margin on one side of convolution corresponding to the external limiting membrane of the retina.
(3) neurological formation.
(4) globular areas filled with vitreous humor.
(5) local invasion of underlying tissue.

Other reports have described pigmented epithelium, islets of cartilage, and hyaline droplets or spheroids in spaces bounded by the cellular membranes. This tumor is by nature identical with brain tumors; thus mesenchymal elements do not constitute an integral part of its growth. The tumor, however, does derive structural support from connective tissue and mesenchymal elements within the eye.

In 1908, Fuchs[5] classified all tumors of the ciliary body and proposed the term diktyoma for this neoplasm. This is a nonspecific term, which, according to Verhoeff,[9] had been applied earlier by Emanuel to the tumor now called retinoblastoma.

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The tumor is malignant, but local invasion of tissues within the eye is usually the limit of extension. Malone's report[7] is unique in that death resulted from systemic metastasis.

CASE REPORT

The patient was first seen at the University Clinics when he was 8 months old. During the seventh month the parents had noted that the iris of the left eye was brownish in color and that the pupil had become opaque. The past history and family history were noncontributory. The mother died of a metastatic chorioepithelioma shortly after the infant was first seen.

Examination showed a normal blue right eye. The left pupil was irregular and did not react to light. An immature cataract was present with posterior synechias at four and at eight o'clock. The iris was atrophic and much darker than that of the right eye. The left cornea was very slightly hazy, and mild ciliary injection was present. The fundus could not be visualized.

A diagnosis of iridocyclitis with cataracta complicata of the left eye was made. Roentgen ray studies of the skull, optic foramina, and chest indicated no abnormality. Skin tests with tuberculin, brucellergin, histoplasmin, coccidioidin, and toxoplasmin were negative. Hemoglobin was 12.4 grams; erythrocytes, 4,900,000; leukocytes, 14,200 with 26 per cent polymorphonuclear leukocytes, 73 per cent lymphocytes, and 1 per cent monocytes. The toxoplasmosis dye test was positive in 1:64 dilution and the complement fixation was negative. Complete physical examination revealed no abnormality other than the eye. Treatment with local cortisone and atropine appeared to cause some reduction of the amount of inflammation.

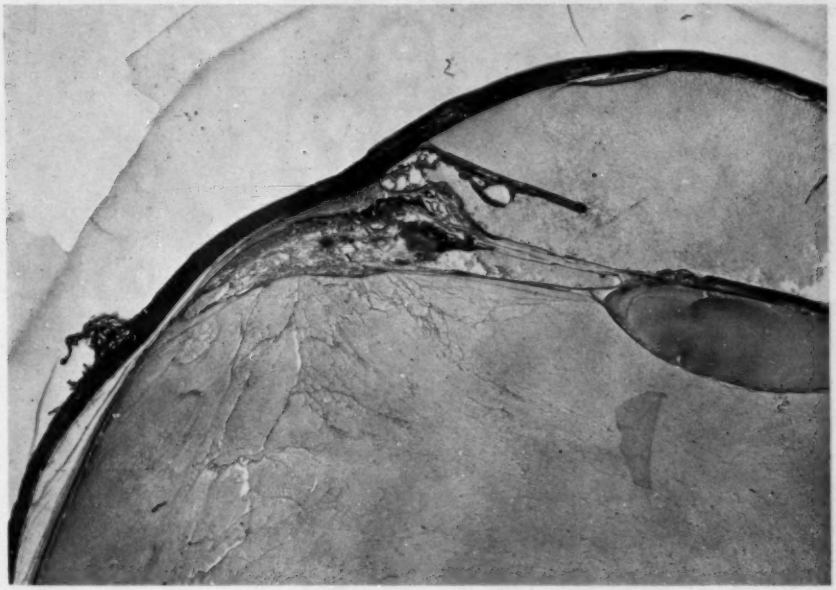

FIG. 1.—Low-power photomicrograph of the diktyoma, which has ruptured the capsule of the lens, obliterated the corneal scleral angle and spread over the posterior surface of the iris. (Hematoxylin and eosin; × 11)

Two months later, when the patient was next seen and when he was 10 months old, the eye was markedly enlarged. The cornea was edematous, the pupil irregular, and the vessels of the iris engorged. The lens was dislocated nasally, and a black mass was present in the temporal portion and did not transilluminate. Intraocular pressure was 39 mm. of mercury Schiøtz. The eye was enucleated several days later without incident. The child has

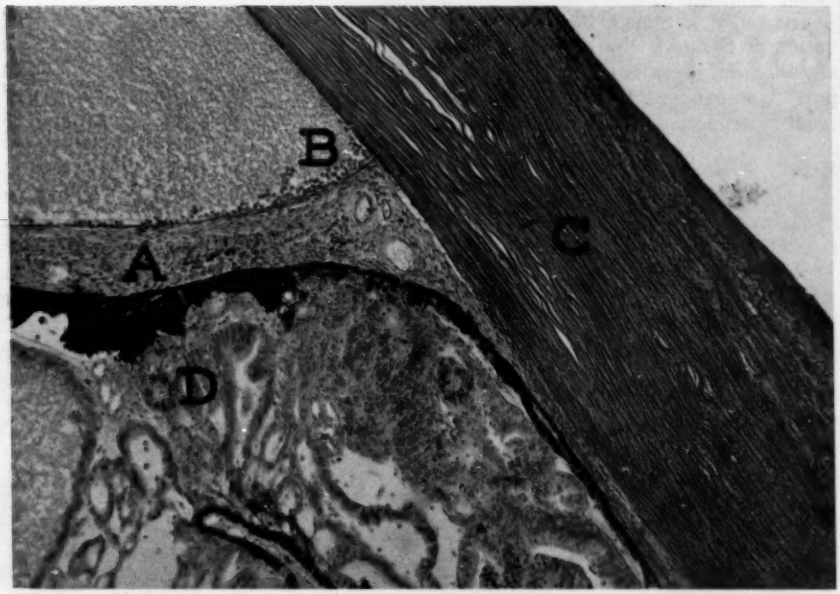

FIG. 2—Cells in the anterior chamber; obliteration of the corneoscleral angle; proliferation of iris pigment and a portion of the tumor. A, iris; B, angle; C, cornea; D, tumor. (Phosphotungstic acid and hematoxylin)

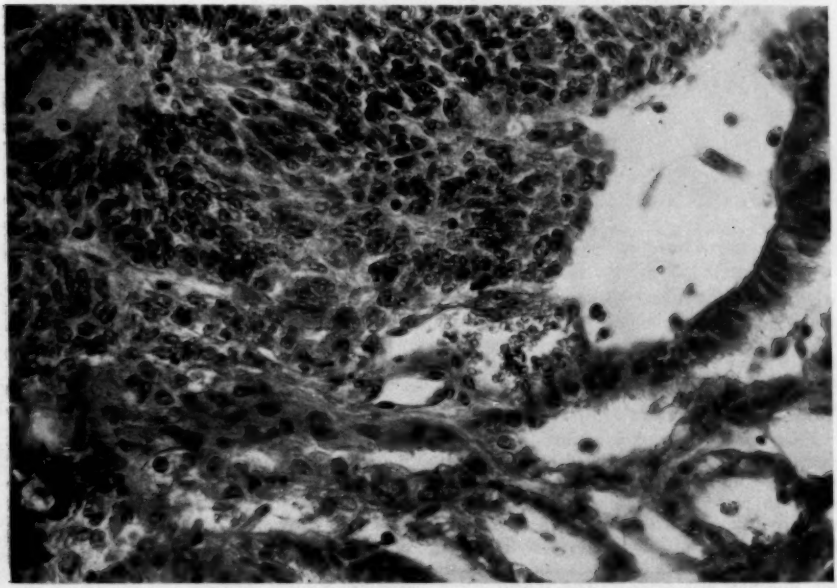

FIG. 3—A typical portion of the tumor with tubules and solid areas. (Hematoxylin and eosin; × 400)

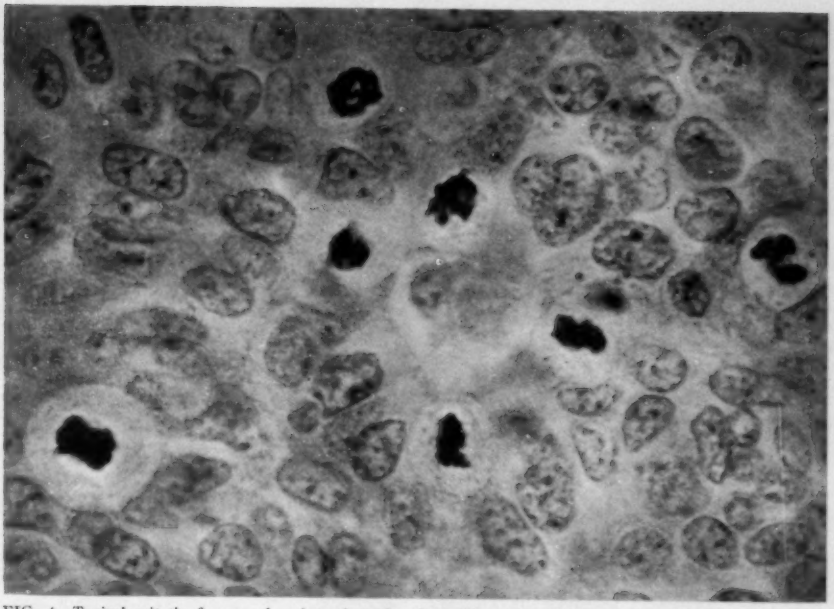

FIG. 4—Typical mitotic figures of embryonic retina in solid portion of tumor. (Hematoxylin and eosin; × 1275)

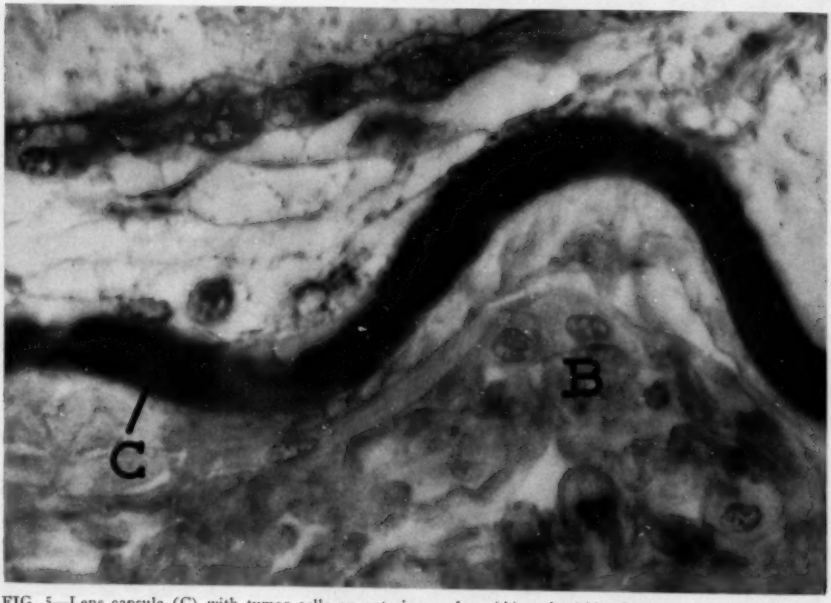

FIG. 5—Lens capsule (C) with tumor cells on anterior surface (A) and within the lens (B). (Van Gieson: × 1230)

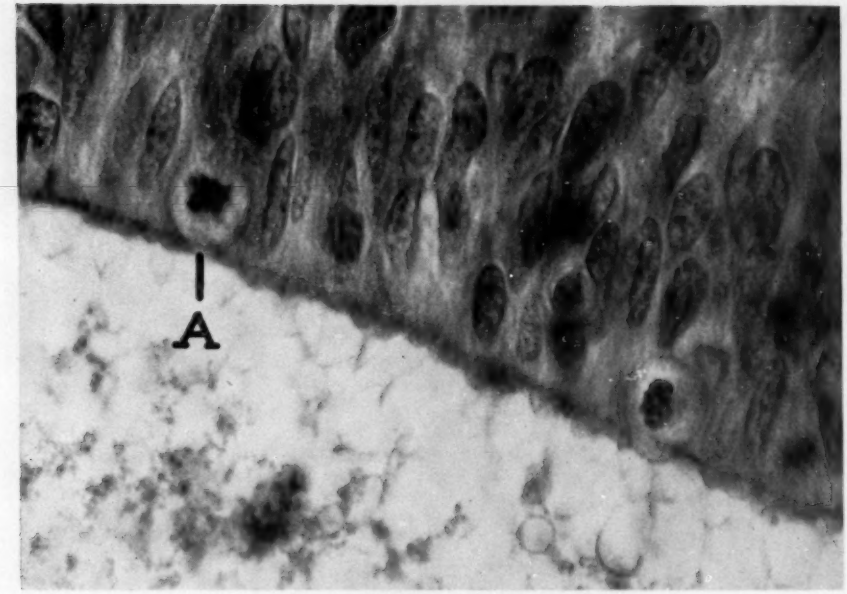

FIG. 6—Attempt to form external limiting membrane (A) by tumor cells. (Van Gieson; × 1135)

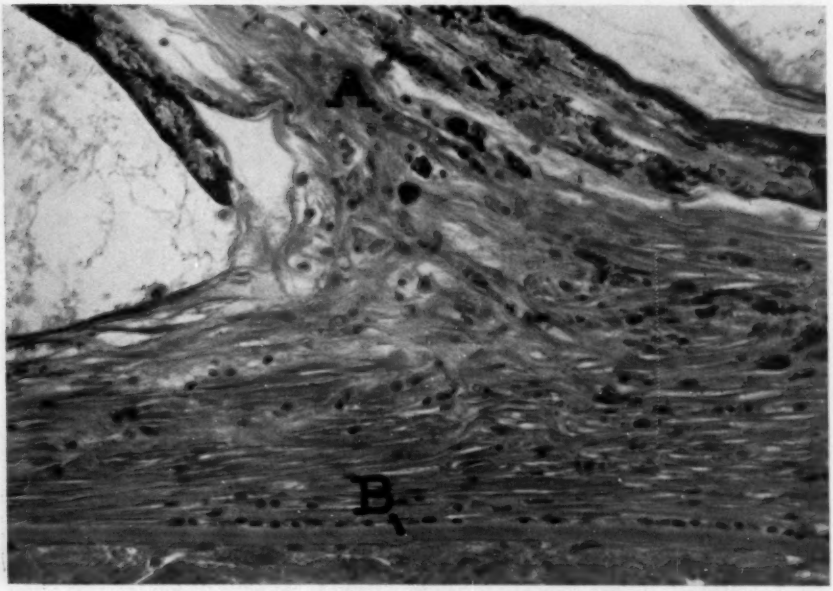

FIG. 7—Iris (A) adherent to lens capsule (B) with much connective tissue formation.

been entirely well since the operation more than a year ago and there is no evidence of local or distant recurrence.

PATHOLOGIC EXAMINATION

The enucleated eye was fixed in Held's solution and sectioned horizontally. Serial sections 13 to 17 microns thick were made, and every tenth section was stained with hematoxylin and eosin. Other sections were stained with phosphotungstic acid hematoxylin, Van Gieson's stain, Weigert's stain, and trichrome stain.

The cornea measured 11 mm. vertically and 12 mm. horizontally. The bulb measured 23 mm. anterioposteriorly and 22 mm. vertically. An area of the globe in the three o'clock (temporal) meridian did not transilluminate. The anterior chamber was practically obliterated, and the iris bulged forward obscuring the angle. Grossly, the choroid, retina and sclera appeared normal.

In the microscopic sections the cornea was essentially normal. The anterior chamber was very shallow and filled with an albuminous coagulum which stained pink with hematoxylin and eosin. Numerous cells, chiefly polymorphonuclear leukocytes, were present in the anterior chamber and lay upon the anterior surface of the iris. The iridocorneal angle was entirely closed by peripheral anterior synechias. There was endothelial proliferation on the iris surface and marked atrophy of the iris stroma.

The pupillary border was markedly everted and overgrown, and there was generalized proliferation of the iris pigment. Broad posterior synechias were present in many areas.

The lens was shrunken, cataractous and dislocated to the nasal side. Numerous bladder cells were present, and there were many clefts in the cortex. The anterior capsule was ruptured near the equator, apparently by traction of the tumor upon the zonule. In this area there were scattered lymphocytes with a few macrophages within the lens substance. Overlying much of the anterior capsule in the area of the rupture was connective tissue containing similar inflammatory cells. The choroid, retina, sclera and optic nerve were normal.

Filling the posterior chamber on one side was a tumor of unusual appearance consisting of tubules, bands, and solid areas. Two main types of cell were present. A low cuboidal type of epithelium predominated in the linear bands of cells which line the zonular fibers, covered some of the ciliary processes, and extended over the posterior surface of the iris. In more solid areas of the neoplasm, these cells were more elongated and more irregular in shape and size. The second type of cell occurred much less frequently and was globular in shape, with a paucity of cytoplasm. A complicated mitotic figure was present in these cells in the central portion. Associated with these cells, which resembled the embryonic retina prior to stratification, was a sharply defined homogenous linear structure resembling the external limiting membrane of the retina.

Many tumors were arranged in long bands which extended over the zonular fibers and onto the surface of the lens but did not invade it even in those areas where the capsule was ruptured. These bands were frequently only a single cell wide, and except for position, resembled the normal nonpigmented epithelium of the ciliary body. In some areas, tumor cells were arranged in large cystlike processes with a homogeneous pink material contained within. In solid areas of the tumor, rosettes of cells were present.

In one small area, the tumor had broken through the pigment epithelium to

involve the ciliary muscle. Other than this there was no evidence of invasion.

Pigment occurred irregularly in isolated portions of the tumor but appeared to be entirely derived from that of the ciliary processes. Connective tissue stains indicated that the tumor was entirely devoid of these elements, but that some of the structural support of the tumor was derived from them.

The pathologic diagnosis was made of diktyoma, rupture of lens capsule, cataract, subluxation of the lens, lens-induced inflammation, and secondary glaucoma.

DISCUSSION

Clinically this patient presented a most difficult problem. At the time of enucleation it was evident that a tumor involving the ciliary body and causing subluxation of the lens was present. In differential diagnosis, the age of the patient tended to exclude malignant melanoma, the absence of skin lesions made the diagnosis nevoxanthoendothelioma unlikely, and the presence of inflammation made a diktyoma or a retinoblastoma unlikely. An inflammatory granuloma thus seemed to be the most likely cause.

The occurrence of inflammation with the diktyoma in this case must be considered most uncommon. Malignant epithelioma, the other malignant tumor of the ciliary body, is usually preceded by intraocular inflammation, and this together with the age of the patient tends to differentiate it from the diktyoma.

Study of the eye indicates that the inflammation occurred because of rupture of the lens capsule. This appears almost certainly to have occurred about the child's seventh month, when first the parents noted difficulty with the eye. The position of bands of tumor cells on the zonular fibers and the presence of tumor cells on the lens cortex itself suggest that the tumor itself was responsible for the capsule rupture and subsequent inflammation.

Secondary glaucoma occurred when the tumor was still relatively small; indeed, this child is younger than the usual patient with diktyoma although the tumor occurs almost exclusively in children. The secondary glaucoma thus resulted from the intraocular inflammation rather than from the increase in size or necrosis of the tumor.

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THE ANATOMIST LOOKS AT THE TEMPOROMANDIBULAR JOINT

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THE ear is the only feature that mars the smooth contour of the side of the human head. The patient is conscious of no other structures between the tip of the nose and the external occipital protuberance; hence, any pain between these two landmarks is assigned to the ear. The otolaryngologist makes the differential diagnosis. Routine examination readily shows whether the ear is the source of pain. Pharyngeal ulcers which give pain referred to the ear are sought. The occurrence of a parotitis is considered. Perhaps the very rare pharyngeal crisis of tabes is brought to mind. If all of these leads are negative, the temporomandibular joint, which has one bony wall in common with the ear, must next be examined.

The dentist and otologist have contributed both to the knowledge and to the confusion about this joint. Concerned as it is with speaking and eating, it is perhaps our most essential joint. It is subjected to more physiologic change than any other. Between toothless infancy and toothless senility, this joint accommodates to the eruption of two sets of teeth and to their subsequent loss. This calls for tremendous adaptation. While we think of bones and joints as rigid, on a long-term basis they are some of the most plastic structures in the body. The series of transformations in this joint occur without any awareness on the part of many of us. On the other hand, with slight wearing of the tooth surfaces, or following extraction of the third molar, some have trouble. Young persons may have pain due to some minor malocclusion. Some of the complaints may be due to an individual hypersensitivity, but other patients may have structural differences. In my opinion, difficulty with this joint is much more common among those persons said to be hyposthenic. These are the people with loose joint capsules and hypotonic musculature. Parenthetically, this may be the basis for the good results obtained in some patients following the injection of fibrosing solutions.

We have noted the great changes that the joint accommodates to in a lifetime. There are several other features that make this joint unique. Each joint is linked to its mate of the opposite side. It is true that there are two condyles on the skull for the first vertebra, and that there are two condyles on the femur which articulate with the tibia. These pairs of condyles are close together, and there is little opportunity for any great disturbance of relationship between a pair. The condyles of the mandible are widely separated, and interposed between them are all of the irregularities introduced by the changes in the teeth. The problems of this joint would be much simpler if teeth erupted and were lost in bilaterally symmetrical occluding pairs.

Each temporomandibular joint is actually two joints. This is stated not to attract attention but to emphasize an essential fact regarding the anatomy of a joint with a disc. Think of a finger with

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three phalanges and two joints: if the middle phalanx should become shorter and shorter, eventually only the two joints would remain with only the cartilaginous ends of the bone between them. It is the anatomists' custom to speak of this as one joint with a disc in it. It is easily shown that every joint containing a disc represents a place at which movement occurs at two surfaces. In the temporomandibular joint, the head of the condyle moves on the disc, or the head of the condyle and the disc move as a unit on the temporal bone. If there is trouble in the joint itself, either portion or both might be involved. Any injections should be made with this dual character of the joint in mind.

The temporomandibular joint capsule, like all joint capsules, has a certain looseness. This looseness is produced by, maintained by, and permits the normal movements of the joint. When the condyle moves forward, the folds are pulled out of the posterior part of the capsule and the anterior portion is thrown into folds. Upon return of the condyle, the reverse takes place. The forward and backward movement of this condyle is extensive. The displacement of tissue which might be expected does not seem to occur. Recently I noted a rich plexus of veins in front of and behind the condyle. This anterior and posterior condylar plexus has been repeatedly confirmed by corrosion preparations and by dissections. The anterior plexus anastomoses freely with the posterior plexus. I saw these vessels some years ago but did not appreciate their significance. This appears to be a hydraulic mechanism which adjusts for the changes in pressure and volume in the tissues (fig. 1). When the condyle moves posteriorly, these posterior veins are compressed and the blood moves into the anterior plexus. When the anterior plexus is compressed by the condyle, the blood rushes posteriorly. This probably explains why so few of us show a dimpling in the preauricular region when we open our mouths.

FIG. 1—Corrosion preparation of the veins about the condyle of the mandible: c, condyle; a, anterior plexus; p, posterior plexus. The connecting network joins the two plexuses.

How wide should we be able to open our mouths? This query receives various answers, but there seems to be a medical-dental old wives' tale that the patient should be able to place the middle phalanx of the middle finger between the incisor teeth. This seems to be one of those legends by which we convince ourselves that we are normal. I cannot recall anyone, with the exception of patients with trismus, who could not open his mouth several millimeters wider than the opening indicated by this criterion. I well remember a soprano singer who, after a jaw infection, could not open her mouth wide enough to reach her three highest notes. When she placed the middle phalanx between her incisors, there

was room to spare. The injection of procaine into a spastic masseter muscle secured an opening sufficient to restore her full scale.

The mouth of the average individual probably opens widest when his physician asks him to say "Ah." Talking does not require much separation of the teeth, and incising and chewing modern food require only a minimal opening (figs. 2 and 3). The amount of opening depends upon the consistency of the food. Most movements of the mandible require little muscular effort. This is the type of muscle contraction generally called isotonic: the muscle changes in length but the tonus remains about the same. When we move the jaw in speaking, when we position the mass of food between the occlusal surfaces, and even when we occlude down to the last degree, we are using muscles in this isotonic sense. When the molars are almost in contact, separated by a thin film of food, the muscle shortening cannot continue; the length of the fibers remains the same; and the contraction is said to be isometric. The tonus increases until the food is milled to the desired fineness. It is under this circumstance that sufficient muscle force is exerted to traumatize tissues. Considering the shortness of our eating period and the character of our food, this does not leave much time to traumatize tissues by mastication. The principle damage seems to be done when the tense individual with the teeth in contact, rhythmically contracts his masseter and temporalis muscles to their fullest extent. It is as if the clenched jaw is endeavoring to keep the tongue from speaking out. I am told that some schools of acting teach their students to contract the temporalis and masseter muscles rhythmically to indicate deep emotion. If the present trend continues, we may have to cease speaking of the seventh nerve as the nerve of facial expression.

FIG. 2—Roentgenogram of the right temporomandibular joint while food was being incised. This patient has little forward movement of the condyle and very little rotation. The incisors separated 20 mm. to bite a 15 mm. biscuit.

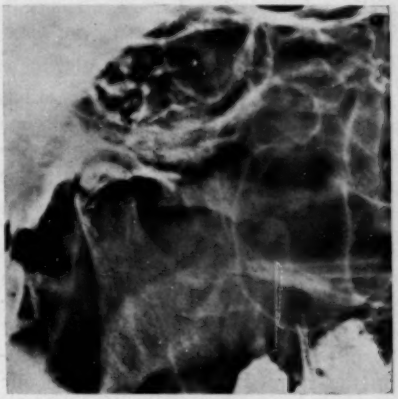

FIG. 3—Roentgenogram of same joint as in figure 2. The subject is chewing. The relations of the condyles are practically the same as in figure 2. These two figures emphasize the small amount of opening required for eating. The amount of protrusion is not related to the extent of the opening.

I believe that the joint is involved most when it is acting as a freely moving hinge. This movement is accomplished without marked muscular effort, and I believe that in the range required in our normal physiology, it is not a traumatic movement. The function of crushing

food can be accomplished without the joint at all, and I believe that normally we do not exert enough force here to justify thinking of this joint functioning in the milling of our food.

Three clinical cases will highlight this this concept. Dr. John W. Conwell[1] of Cleveland resectioned parts of several mandibles for mandibular cysts. After this resection the patients seemed to have adequate function of the mandible. Dr. Conwell was kind enough to record one of his cases for me on a motion picture film. Careful study of this film does not seem to show any essential changes in function, and roentgenograms of the remaining joint are quite normal. On the left side, this patient has no mandible posterior to the second bicuspid (fig. 4). She has to masticate with her anterior teeth only because she has no occluding posterior teeth on the side not operated upon.

FIG. 4—From the motion picture of Dr. Conwell's case. Excellent function was present after resection of the left side of the mandible from the second bicuspid posteriorly.

Dr. Victor Frank of Philadelphia has allowed me to study and photograph one of his patients who had a similar resection at the age of eleven years. The patient is now twenty-one years old, and his mouth condition is much better than that of the last patient. He carries out a normal round of activities in which he is constantly before the public as a teacher. He has no embarrassment because of any peculiarities of mandibular movement. From these two cases one might assume that both joints could be dispensed with. This is what is done with patients with ankylosis of both condyles. These cases, however, are not ideal cases for study because the mouth is so anomalous that competent conclusions are difficult.

I have had the opportunity to study a most informative third case through the courtesy and cooperation of Dr. Arthur E. Smith[6] of Los Angeles. The patient was in an automobile accident which caused extensive fractures, including fractures of the necks of both mandibles. Dr. Smith removed the condyle bilaterally. The early results were excellent and were shown at a clinical meeting in Los Angeles. As this patient was observed at dinner, it was obvious that she had excellent dental function. The patient moved from the Los Angeles area, and it was not until sixteen years later that Dr. Smith[5] located her again. She was now definitely a dental cripple. A marked recession of the mandible was present, and great separation of teeth in the molar area was evident. The patient had difficulty in chewing and did not like her appearance. This case history ends on a happy note, since Dr. Smith reconstructed these joints, restored the patient to dental health, and improved her appearance.

It is now clear that the loss of one temporomandibular joint does not disable the patient. Both condyles can be removed, and with proper care, the early results (at least for 18 months) are excellent. However, from the long-term aspect, the condyle and the joint are necessary to maintain proper occlusal relationships.

All of this presents a point of view different from the usual one about this joint. I feel that the temporomandibular joint acts like a hinge, with very little muscular effort, in speaking and in positioning food for mastication. I believe that it functions as a structure maintaining the occlusal relations of the teeth by preserving the anterior-posterior relationship. One joint seems to be adequate for this latter purpose.

This brings us to the problem of pain in the joint area, particularly the pain radiating along the auriculotemporal nerve and generally called auriculotemporal neuralgia. I shall not discuss certain pains, deep in the face, hard to deny, and difficult to confirm. I shall not suggest that we do not have pains which follow the blood vessel pattern. These are not as important as the pain which radiates along the anatomic course of the auriculotemporal nerve, from in front of the ear in a fan-shaped manner to the vertex. By anatomic coincidence, this nerve takes a course behind the condyle and neck of the mandible in the soft tissue. Professor Harry Sicher[4] has frequently insisted that change in the position of the condyle after the loss of teeth will not traumatize this nerve. Dr. Sicher prefers to think of this pain as of reflex origin. It is not necessary to settle the genesis of the pain. The important point is that this radiating pain can be relieved by opening the bite. The diagnosis is readily made. With the two index fingers just in front of the ear and behind the condyle, the patient is asked to open the mouth widely. At this point, the examiner uses his index fingers to force all of the loose tissues possible behind the condyles (fig. 5). The patient is then told to close the mouth. To some patients this gives no discomfort; most patients will cringe. If the patient has pain, either he will tell you, "It hurts, but that isn't the pain I'm talking about," or he tells you immediately, before he gets the jaws into occlusion, "That's the pain I'm talking about." If the patient gives this last response, you may be sure that you are dealing with auriculotemporal neuralgia. You can now extemporaneously open the bite by putting almost anything between the occluding surfaces to separate them. If the patient now occludes with force, you cannot bring back the pain by pressing behind the condyle. A peculiar fact is that after you remove the substance from the occlusal surfaces, the patient can open and close the mouth once or twice without the occurrence of the pain when pressure is exerted on the retrocondylar area. I assume that this is due to some rearrangement of the loose tissue of the joint capsule. From this point on, the problem is one for the dentist. Will he be able to open the patient's bite sufficiently to eliminate the auriculotemporal neuralgia? He generally can. Let it be emphasized that it is not necessary for the patient to wear appliances for several months to determine whether relief can be obtained. The diagnosis can be made and the treatment tried out extemporaneously, as above. The patient can be assured, if the extemporaneous results were satisfactory, that the den-

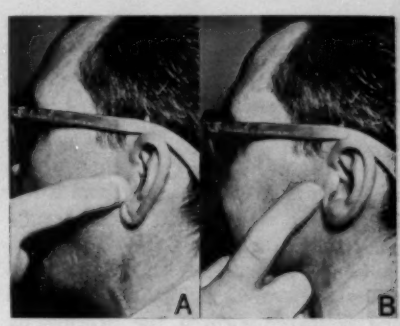

FIG. 5—Testing for auriculotemporal pain due to joint changes accompanying a closed bite. (A) the index fingers are placed just behind the condyles and the patient is told to open the mouth fully; (B) the index fingers push the loose tissues behind the condyles and the patient is told to close the mouth. This maneuver precipitates the pain typical of a closed bite.

tist in all probability can eliminate the trouble. There is a rare case in which the bite cannot be opened sufficiently.

Earlier, the problem of deafness was associated with auriculotemporal neuralgia. This now has no competent support, but patients still ask about it and a few dentists still claim results. After several reports in the dental literature, Dr. James B. Costen,[2] in 1934, reported a series of 11 cases in which he believed that he had improved the hearing by opening the bite. Remember that audiometers were not in common use. In 1942, Dr. Costen[3] with great honesty said that he could find no causal relationship between the closed bite and loss of hearing. That is where the problem stands.

I have discussed pain along the auriculotemporal nerve which can be relieved by opening the bite. Arthritis in the temporomandibular joint has to be treated as arthritis is treated elsewhere. I do not believe that the removal of a condyle, or converting what is normally two joints into one joint, is a logical procedure. If you go through the simple diagnostic maneuver of pushing the soft tissues behind the condyle as the patient forcibly closes the mouth, the resultant pain will confirm a diagnosis of auriculotemporal neuralgia. If the patient is relieved by extemporaneous opening of the bite, good clinical results may be expected from dental treatment.

Photographs by Mr. Alex Vaskelis.

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DISCUSSION

JAMES B. COSTEN, M.D., St. Louis: It is gratifying to discuss Dr. Batson's paper, mainly because he has the practical anatomic approach. His interest in the functional anatomy of the temporamandibular joint covers a great many years. My first clinical studies had just begun when he made valuable suggestions to me regarding points which should be cleared in order to explain the symptom complex, and his criticism has always been constructive. That is a very important thing about everything Dr. Batson does. You rarely, or never, hear him utter destructive comment; he always points his remarks toward what we must do to proceed.

You have heard a clear analysis of the tonus values of the masticator group of muscles: the temporalis, the internal pterygoid, and the masseter muscles. The gradual reduction of vertical dimension follows abrasion and loss of teeth, alveolar shrinkage followed by changes in the temporomandibular joint. No pain is experienced unless a trismus factor is introduced. If any local source of pain occurs, such as impaction of the condyle from a blow on the chin, stretching under anesthesia, or inflammatory reaction to third molar extraction, sensory stimulation on that side occurs. The masticator muscle group contracts, and trismus of this homolateral group of muscles is produced as long as sensory stimulation remains. The condyle exerts more than normal pressure, and destruction of the joint structures is more rapid than the slow progress of advancing age.

Actually, trismus is the simplest title, for this function is invariably present in some stage of destruction of the joint unless a paralysis of one side leaves the normally functioning muscle in a relatively greater state of tonus.

"Auriculotemporal neuralgia" is the correct term for the part of this condition related to irritation of the auriculotemporal nerve or its branches, but "mandibular joint syndrome" was chosen as a title for my studies for two

reasons: (1) to avoid confusion with the auriculotemporal syndrome of vasomotor disturbance after seventh nerve paralysis; (2) to include other possible sources of pain about the joint, such as the chorda tympani, later shown to carry pain fibers. In the study on pain fibers, on which I worked for two years with Dr. Bishop, we found that the largest touch fibers were 12 to 15 microns, and pain fibers were 5 to 6 microns. Referring to Gasser's and Erlanger's original studies on the great nerve auricular, we determined that the taste fibers were 2 to 4 microns. On this basis, it seems evident not only that there are pain fibers in the chorda tympani nerve but that there is a preponderance of pain fibers in that nerve. If Dr. Batson will admit the occasional possibility of irritation of that nerve by some destruction of the joint and medial shift of the condyle, then we can include the chorda tympani as a source of pain as well as the reflex action from the auriculotemporal nerve. The articular branch of the temporal artery was assumed by Horton, Magath, and their co-workers, and by Profant, to be the origin of painful temporal arteritis.

Dr. Batson's description of testing for mandibular joint pain by positive muscle action on closure packs is interesting. My procedure, which is based on the deduction that this pain, if present, will be relieved by fixation or movement of the condyle, depends on passive splinting of the jaw. I apply an elastic bandage to the chin for short half-hour periods, at the same time increasing slightly the intramolar space on the side opposite the pain. This works well in most cases, the exceptions depending on more complicated jaw and joint changes.

Assuming that the diagnosis of temporomandibular joint disease is correct, reliable dental records indicate that about 15 per cent of these cases fail to respond to treatment. Suppose that we do not call these dental failures; in return the dental section should refrain from stating, "The occlusion is balanced, the symptoms remain; therefore, the theory is not good!"

First, an arbitrary fraction of these failures must be reclassified as cases of conversion neurosis. These patients either have no mandibular joint changes or have developed a neurosis which they refuse to give up. Others may be characterized by a strong painful crepitus, the Kiefergelenkknacken of Axhausen. This is the arthritic change referred to by Batson. These patients may be made comfortable for periods of one to six months by injection of hydrocortisone directly into the joint or, when the pain is uncontrolled in this manner, by surgical removal of the disc, to which Batson referred. When we do this occasional external correctional procedure, bony projections, or exostoses, are noted about the condyle as well as actual erosion of the anterior face of the condyle, leaving a roughened, sharp-edged surface to snap against the adjacent tissues.

We have been conservative in this country in using the direct surgical attack, and Dr. Husted, of Copenhagen, informs me that he is becoming more and more conservative. Only a few years ago, in his book "Temporomandibular Arthrography," Norgaard referred to 37 cases, in 25 of which he did a surgical procedure.

There still remains unexplained a fraction of the 15 per cent of cases not cured by dental treatment. During the past two years, with the support of the John Swift Fund, we have observed the phenomenon of isolated masseter tremor. Complete neurological studies were made, ruling out Parkinsonism, senile tremor and bulbar palsies. By direct palpation of the masseters, a tremor of three cycles per second was found; it faded under pressure, but was present again when palpated. Cases of isolated masseter tremor are rare. In these cases no relief whatever is obtained from occlusal or joint treatment. The prognosis is poor, but since these tremors have a functional source, the patient is very discouraged when told that nothing can be done. Actually, passive support by external elastic splinting is more effective than any other treatment that I have used.

To quote Dr. Batson further, "Hearing loss is no longer considered a part of the syndrome." We must be reminded that "ear symptoms," as studiously written into the original title, belong to the syndrome, for a large proportion of patients have otalgia and honestly believe that their hearing has been improved whether we can show it with audiograms or not. In tabulating the symptoms reported in a large number of cases, otalgia was found to be the commonest complaint. When relieved by occlusal restorations, the patients insist that they hear better; yet when patients were grouped as to age, background disease and otological history, and when serial audiograms were made, consistent improvement in hearing was never found which could be attributed to relief from occlusal difficulties.

JOHN B. ERICH, M.D., Rochester, Minn.: I want to compliment Dr. Batson on a very interesting and informative presentation. There is little that I can add, but I believe that at present far too many operations are performed on the temporomandibular joint.

The most common disorder of this joint is a traumatic arthritis, characterized by pain, partial trismus, and clicking and grating during movements of the joint. It is our belief at the Mayo Clinic that this condition seldom warrants surgical removal of the meniscus and never warrants condylectomy.

One reads glowing reports in the literature concerning the complete relief of all symptoms following the removal of the meniscus. However, the beneficial results of such operations too frequently are not permanent. As a matter of fact, many patients who have had the meniscus removed come to our clinic seeking relief from the previously mentioned symptoms, which have returned in spite of the meniscectomy. Furthermore, we are encountering more and more patients with traumatic temporomandibular arthritis who have had not only the meniscus removed but, subsequently, the condyles as well. Such radical surgery seems unwarranted and occasionally may lead to complications such as partial temporamandibular ankylosis. As Dr. Batson pointed out, bilateral condylectomy not infrequently produces an open bite which seriously disturbs the patient's ability to incise and masticate food and which can be only partially corrected by an extensive amount of dental work.

Unless traumatic arthritis of the temporomandibular joint is associated with ankylosis, I can see no reason for ever performing a condylectomy in these cases. In our experience, symptoms of this disorder usually disappear in a matter of days or weeks if disturbances in dental occlusion are corrected and if the patient is instructed not to open his mouth too wide. I agree with many surgeons who believe that the meniscus should not be removed unless the patient has had severe pain for several months.

One factor which augments the severity of the patient's symptoms and which has received little attention in the literature is fear and worry on the part of the patient. Many individuals fear that the condition is the beginning of a generalized infectious arthritis. Others worry for fear their jaw joints will become completely ankylosed. I have found that, when such patients are assured that these complications will not occur and that their symptoms eventually will disappear, they usually have no further difficulty in enduring the discomfort of this disorder.

We have cared for a great many people with traumatic temporomandibular arthritis who live in the immediate vicinity of Rochester and who come in for observation at frequent intervals. We never have removed the meniscus from any of these patients, and none have failed to recover from the painful symptoms of this disorder. Some, however, have had occasional recurrences of symptoms at varying intervals.

I do not believe that disturbances of the temporomandibular joint associated with faulty dental occlusion account for a burning tongue. Burning of the tongue is largely a functional complaint. It seems that almost any form of therapy to the jaws on a patient with this paresthesia of the tongue may relieve the burning sensation.

Lastly, I would like to discuss very briefly fractures of the neck of the mandible, so-called subcondylar fractures. Many surgeons believe that fractures of the neck of the mandible with dislocation of the condyle and complete disruption of the temporomandibular joint require an open operation in which the condyle is either replaced and pinned in proper position or else removed entirely. In my experience, such operations not only are unnecessary but can be attended with complications, such as disturbances in dental occlusion and partial fibrous ankylosis. Moreover, repositioning and pinning a condyle occasionally can cause resorption of the whole condyle.

We have treated a great many patients with subcondylar fractures and have undertaken no operations to reposition or to remove the condyle unless the condyle was driven into the external auditory canal. Instead, treatment consisted merely in fixation of the teeth in proper occlusion for a period of not more than 3 weeks. Regardless of displacement of the condyle, we never have had a patient who has had any permanent difficulties with his jaws. There is always, of course, the potential but remote possibility that in a child with a subcondylar fracture a bony ankylosis of the temporomandibular joint or a disturbance in the growth of the mandible may eventually develop. However, I am convinced that these complications would occur whether or not an open operation on the joint were performed at the time of the injury.

Symposium: Errors in Early Diagnosis and Treatment of Cancer in the Upper Respiratory Tract

THE EAR

J. E. MAGIELSKI, M.D.
ANN ARBOR, MICHIGAN

PHYSICIANS dealing with cancer of the ear realize that there will be less chance for error in making a diagnosis of lesions affecting the auricle and external auditory meatus than there will be among the tumors which occur in the proximal external auditory canal, middle ear and mastoid. Discussion will therefore be directed to this latter region of the ear.

Perhaps the most conspicuous error is a lack of a high degree of suspicion of cancer on the part of the examining physician because the disease is not common. At the University of Michigan Hospital, carcinoma of the external auditory canal, middle ear and mastoid occurred in one patient out of 8,900 attending the clinic. Cancer in this location occurred in only one patient out of 280 patients with chronic suppurative otitis media and mastoiditis. Reports of surveys in other clinics indicate that the incidence may be even lower.

Since there are no characteristic early symptoms of cancer of the external auditory canal, middle ear and mastoid, the failure to obtain a detailed, chronological and adequate history of the patient's illness may lead to an error in diagnosis. However, one constant feature of the illness is a purulent foul aural discharge of less than a year's duration in most instances. This point in the history must be related to the course of the illness, since this disease is easily confused with a chronic suppurative otitis media and mastoiditis. The discharge may be insidious in onset, and later it may be associated with pain out of proportion to the physical findings. A history of persistent bloody aural discharge is particularly important, especially in a person over the age of 40.

If errors in the early diagnosis of cancer are to be avoided, a painstaking examination of the ear is mandatory. The external auditory canal must be thoroughly cleansed to ensure maximum visualization of the canal and tympanic membrane. A pneumatic otoscope can be of great value in studying the relationship of the eardrum to a lesion in the canal or in the middle ear. Palpation by means of a silver probe under direct vision as an aid to inspection must not be forgotten. One may detect friable, easily bleeding granulation tissue or the presence of denuded bone by this method. Either of these findings should serve to arouse the suspicion of the presence of carcinoma. Failure to suspect carcinoma when any form of "granulation tissue" is present in the ear and failure to examine microscopically all tissue removed from the ear are grave errors. In children this is especially true, since sarcomas of the ear may be rapid in growth and appear as large aural polyps with a

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history of suppuration of short duration. If cancer is suspected, one must persist in pursuing a diagnosis of cancer even though the first biopsy report is negative. Such an error can occur, since exuberant granulations may develop as the result of secondary infection and thus disguise an underlying malignant lesion.

Another error which may delay diagnosis is the indiscriminate prescription of topical or systemic antibiotics for the patient with a discharging ear. Again, secondary infection is a common feature of a malignant lesion in the ear. The indiscriminate use of antibiotics can result in partial temporary decrease in the patient's symptoms and may mask the basic cause for his complaints. During the past year we have seen three instances of lipid histiocytosis and one case of carcinoma which had been initially treated in this manner. Early histopathologic examination instead of early antibiotic therapy would have established the correct diagnosis without loss of time.

Too great or too little reliance upon radiographic study of the temporal bone can contribute to a postponement in diagnosis of cancer of the ear. The physician should not let himself develop a false sense of security by acceptance of a report on roentgenograms of "no evidence of bone destruction" as a conclusive finding which rules out carcinoma. Early cancer generally fails to manifest itself by bone changes except occasionally. If the tumor has produced extensive changes visible in roentgenograms, it will usually also be manifest in the middle ear. The roentgen findings should be correlated with the clinical course, and errors can sometimes be avoided by personal consultation and review of films with the roentgenologist. One may err by not obtaining roentgenograms of the mastoid. Consider that group of diseases known as lipid histiocytosis which may be present in the mastoid without early invasion of the middle ear or external auditory canal. Radiographs here are of value in leading to an early diagnosis. Use of roentgen diagnosis may be of utmost importance, especially in children who develop a facial paralysis. Idiopathic Bell's palsy occurs in childhood; but rarely, a primary malignant lesion of the middle ear or mastoid may be responsible for the paralysis, and even though the objective examination of the ear is not revealing, the radiographs may lead to the correct diagnosis.

The management of the patient with cancer of the ear cannot be outlined categorically. In our experience, delay in diagnosis is the largest single factor affecting therapy. The extent of the disease and the nature of the tumor will determine the therapeutic program. In general, radical surgery should be employed whenever possible. Often surgery is combined with postoperative external irradiation or with radium placed into the mastoid cavity. In the event of an extensive surgically inaccessible cancer which is very anaplastic, one may choose irradiation therapy alone.

Let it be said that despite radical surgery with or without irradiation, the prognosis of cancer of the proximal external auditory canal, middle ear and mastoid is very poor. The place of newer forms of irradiation therapy is yet to be determined, but they may possibly contribute to more effective management of these cases. At present it would seem that a vigorous effort toward early cancer detection in this area is required if survival rates are to be improved upon.

DISCUSSION

JOHN B. ERICH, M.D., Rochester, Minn.: I want to agree with and emphasize one thing that Dr. Magielski said. Early cancers of the

middle ear and mastoid produce no symptoms that are characteristic of malignant disease; consequently, if we are going to diagnose these cases early, it will be done only if we keep the possibility of malignant disease constantly in mind and do a biopsy on every abnormal piece of tissue that occurs in the ear canal, especially around the drum.

One type of tumor that I think should be considered along with malignant disease of the ear is chemodectoma. These tumors are not really malignant, but they are often confused with cancer both grossly and microscopically. Of course they are not actually malignant, but they do invade and can kill by entering the cranial cavity. I think that whenever one sees abnormal tissue in the ear canal that bleeds profusely just on touching it, and when this is associated with pain and deafness and discharge, we should think of the chemodectoma. I, for one, believe that surgery does not work out too well in many of these cases. It is very difficult. The bleeding is profuse. At the Mayo Clinic, we have found that irradiation seems to work the best. It reduces the size of the tumor. It reduces its vascularity and tends to keep it from growing.

Symposium: Errors in Early Diagnosis and Treatment of Cancer in the Upper Respiratory Tract

THE NASAL SPACES AND SINUSES

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BECAUSE of the close proximity of the nasal cavities and accessory sinuses to the cranial cavity and orbits, the early diagnosis of malignant tumors in the nose and sinuses is of the utmost importance if satisfactory results in treatment are to be obtained. While it is true that many small cancers in these regions can be overlooked unintentionally because of technical difficulties in visualization, one cannot disregard the fact that there are many errors in the early diagnosis of these tumors which could be avoided and which cause unnecessary delays in treatment.

The following errors in the early diagnosis of malignant tumors in the nasal cavities and accessory sinuses are most significant:

1. Placing too much emphasis on the symptoms of malignant disease in the nose. Although malignant tumors may develop more rapidly than those of a benign nature, they can produce identical symptoms. As a matter of fact, there are no unfailing signs or symptoms produced by malignant lesions in this region; this fact should stress the need for a microscopic examination of every abnormal mass of tissue in the nose or sinuses.

2. Delaying treatment because there are no roentgenographic changes that are suggestive of definite malignant destruction in the bone. The ideal time for treating a cancer in the nose or sinuses is before the development of demonstrable malignant destructive changes in the bone on roentgenologic examination.

3. Removing polypoid masses in the nose without obtaining histologic examination of the tissue removed. One often finds that papillomas, fibromas, plasmacytomas, hemangiomas, pyogenic granulomas and some high-grade carcinomas can simulate polyps.

4. Considering a diagnosis of myxofibroma, chondroma or spongy osteoma as final when the pathologic report is based on only one histologic section. In too many instances, the bulk of such tumors is benign, but interspersed throughout the lesion are foci of serious malignant elements such as fibrosarcoma or osteogenic sarcoma. Consequently, the diagnosis of cartilaginous or osseous tumor should not be considered final until multiple or perhaps serial histologic sections have been prepared. I have found that many of these benign cartilaginous or bony tumors which contain malignant elements can be abolished if radical treatment is undertaken without delay.

5. Considering a negative report from one biopsy as final when clinically a neoplasm is suspected of being ma-

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lignant. Too often an ulcerated or infected malignant tumor will produce excessive amounts of granulation tissue. A single section taken for biopsy may not be deep enough to include the actual tumor cells but may show only granulations or inflammatory tissue.

Other factors which contribute to delays in the early diagnosis of cancer of the nose include (1) procrastination on the part of the patient in seeking medical attention, and (2) carelessness or neglect in examining the patient's nose. Too often, the nasal cavities and sinuses are not examined thoroughly enough to reveal an early malignant process.

THERAPY

Unfavorable results in the treatment of malignant tumors of the nasal cavities and accessory sinuses may be attributed to the following factors:

1. Delays in the diagnosis of malignant tumors until they are too extensive. (This phase of the subject has been discussed previously.)

2. Inaccessibility of some of these lesions.

3. Treatment that is inadequate by necessity because of the extremely close proximity of some of these tumors to the cranial cavity.

4. A tendency to undertreat some of these neoplasms in an effort to avoid facial deformities or injury to an eye.

5. Unsuitable forms of treatment.

DISCUSSION

DR. REED: I should like to add one point. I believe that we have a tendency to forget that the nasopharyngoscope can be used in examining the nose equally well as in examining the nasopharynx. The many areas of the nose proper which are inaccessible to view with the nasal speculum alone can be easily seen with the small nasopharyngoscope used primarily to examine the nose itself.

DR. MILLER: I would like to make just one or two points. One thing has been very interestingly and dramatically pointed out to me with regard to the importance of making a biopsy of all polyps. I was a little reluctant to ask for a routine biopsy of every polyp that was removed until our pathologist stated that he had gone through all of the polyps that had been sent to his laboratory and had found in 3 per cent of the cases, malignant changes which had not been suspected clinically.

I would like to make one point in regard to therapy. A search some seven or eight years ago at a very large community hospital revealed that there were no five-year cures of moderately advanced carcinomas of the sinuses that had been treated by surgery alone or by irradiation alone. Our first cures began to appear when we immediately followed the most radical surgery with complete cancericidal doses of roentgen rays.

Symposium: Errors in Early Diagnosis and Treatment of Cancer in the Upper Respiratory Tract

THE PHARYNX

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PROBABLY the most common source of error in diagnosis of early cancer of the pharynx lies in the nasopharynx. I am sure that you are all familiar with the frequency with which small lesions in this area go undetected. This is mainly due to the relative inaccessibility of the nasopharynx. It is not inaccessible to us as otolaryngologists, however, and by the proper use of mirrors and nasopharyngoscope, we can and must visualize the nasopharynx in every routine examination. I emphasize the need of this in routine examination because the cure rate for nasopharyngeal lesions is almost directly proportional to the time of detection, and symptoms do not usually appear until the lesion is fairly well advanced.

These early lesions are difficult to detect. Therefore, we must carefully explore every bit of the nasopharynx, remembering that lesions here tend to be submucosal and may appear as only a slight swelling or a small crack in the mucosa.

Now, let us get on to the pharynx proper. The very fact that the pharynx is easily accessible gives us a false sense of security because we expect lesions to be easily seen during our routine examination. Small lesions have just as many places to hide in the apparently easily accessible area of the pharynx as they do in the relatively inaccessible nasopharynx.

We are also easily lulled into a false sense of security by the fact that the throat is an area of rather common nonspecific complaints, such as the common everyday lump in the throat, sore throat, and scratchy throat. Unfortunately, early or small cancers of the pharynx have a paucity of symptoms, and if there are symptoms, they may be the same as those seen in these noncancerous complaints. When one sees many such cases in which symptoms are due to these nonspecific causes, it is hard to keep in mind that a small number of these patients may have a small, not easily detectable, cancer.

We must remember to examine the pharynx and the mouth without instruments. In the ordinary examination, the doctor usually pops a tongue blade into the patient's mouth as soon as it is open, forgetting that a small lesion of the tongue can be very effectively concealed from view by the tongue blade itself.

Along these same lines, I'm afraid that we otolaryngologists frequently become so conscious of carcinoma of the larynx, especially in the presence of hoarseness, that we have a tendency to take a quick look at the throat, using a tongue blade, and then proceed to an examination of the larynx. This, of course, is unfortunate because we are becoming increasingly aware of the frequency of multiple primary cancers. Reports in the literature have indicated that between 6 and 11 per cent of patients with cancer have multiple primary cancers.[2,3] So, we must always

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keep in mind that the mere fact that the patient has a nice big carcinoma of the larynx by no means excludes the possibility of cancer elsewhere in the upper respiratory tract.

Probably the most common error committed by otolaryngologists in detecting early cancers of the pharynx is the failure to realize the importance of palpation. You have all heard the old saying that "fingers were made before forks"—well, "fingers were also made before laryngeal mirrors." I will grant you that patients, as a rule, do not take it kindly when the doctor plunges what they think is his whole hand into their mouth while palpating their pharynx and tongue. But, what of the small cancer in the substance of the tongue with no surface changes? This is detectable only by finger palpation, and the same patient would take even less kindly to the doctor who misses this early carcinoma just because he does not want to subject the patient to finger palpation. Needless to say, of course, the finger and the patient should always be protected by the use of a finger cot or glove. Finger palpation should be a routine part of every examination in which there are unexplained pharyngeal symptoms.

The base of the tongue and vallecula is a favorite hiding place of small early cancers in the pharynx. It can be seen only by the use of a laryngeal mirror, and here again we must avoid the tendency to look past the pharynx and the base of the tongue in our anxiety to view the larynx.

The first sign of cancer of the pharynx, or nasopharynx, may be the unexplained swelling of cervical lymph nodes. Primary carcinoma of cervical nodes is rare, and any unexplained enlargement of a node demands a most careful and minute search of the pharynx and upper respiratory tract for a primary lesion before we consider biopsy of the node. It is also important to remember the frequency with which crossed metastasis occurs from lesions of the pharynx.

As you all know, lymphomas are fairly common malignant lesions of the pharynx. These can be very deceiving lesions because they rather quickly undergo secondary infection. The resultant tissue breakdown resembles infection and behaves like infection because such a large portion of the lesion is infection. Results of histopathologic examination of such a lesion are frequently not indicative of lymphoma because the biopsy specimen is taken from the broken down, infected area of the lymphoma rather than from the edges or deep below the infection, where the primary trouble really is.

This then brings up the question of such histopathologic examinations. Despite repetition, I have to stress the importance of not accepting negative findings as final. Many lesions require frequent examination before a positive finding is finally obtained.

We otolaryngologists should also make more use of cytologic examinations for the detection of early tumors. I think that we have lagged far behind other specialties in this regard. There is no excuse for overlooking this valuable means of early diagnosis in our field, in which good cytologic specimens are so easily obtained.

The last common source of error—one that I'm sure we are all frequently guilty of when we are busy—is a failure to take advantage of sufficient local anesthesia when necessary. In many cases it is impossible to examine the pharynx or nasopharynx adequately without the use of local anesthesia. If we are to practice good medicine, we must force ourselves to take the time, at any expense, to obtain anesthesia in these patients.

Now, as to treatment. As we all know, the ultimate prognosis in lesions of the pharynx, whether they be detected early or late, is poor. This very fact, I'm afraid, gets us off on the wrong foot in the field of treatment because it is so easy to develop a defeatist attitude right at the start. A certain percentage, although it be small, of these patients is going to be cured, but the only way to keep the percentage as high as possible is by a vigorous attack on the disease. Every patient should be treated hopefully and energetically as though he were going to be one of those who are "cured."

Treatment of lesions in the pharynx and nasopharynx (excluding the hypopharynx, which really belongs in the province of the larynx) resolves itself into radiation of the primary lesion and surgery or radiation of the metastatic lesions.

Although actual radiation therapy is largely a matter for the radiologist, we must remember that the patient is still our responsibility and that we must see to his over-all treatment and management both as to complications of radiation and as to long-term treatment. It is our job to see that the patient is treated by a competent radiologist who will use adequate doses of radiotherapy.

In choosing a radiologist, I would favor the rare man who has taken the trouble to master the use of the head mirror and examining mirrors. The intelligent administration of radiation therapy is influenced by the progress of the lesion, and the radiologist who is able to observe the tumor by mirrors is better qualified to manage the radiation.

I also feel that we should take advantage of supervoltage therapy and rotation technique, if available. Supervoltage therapy has not shown better cure rate than regular 200 KV therapy, but it is much easier on the patient in terms of skin reactions and secondary reactions.

If the patient has a recurrence after one course of radiation, we should not throw up our hands in defeat; many patients can tolerate two or three courses of radiation. Also, many patients who cannot tolerate further roentgen radiation can tolerate the more localized effect of interstitial implantation of radium needles or radon seeds.

In the matter of metastasis, I feel we must be more energetic than in the past. By and large, metastasis occurs along the cervical lymphatics, and although the over-all cure rate of the primary lesions may be poor, I think that if there is any reasonable chance of cure the patient should have the advantage of a radical neck dissection. I also feel that, in every case, the neck dissection should be complete. If the operation is worth doing, it is worth doing well.

We must be careful not to fall into the trap of thinking that the chances of curing the primary lesion are so poor that we will "give some radiation" to the neck node. The possibility of eradication of cervical metastasis by radiation is very slim, indeed. This should be reserved for palliation in those patients in whom the metastasis is too far advanced for surgical resection.

What of the patient in whom cervical metastasis is present at the time the primary lesion is discovered? The time-honored answer to this problem has been to "include the node in the field of radiation." This, of course, is admitting defeat right at the start because deep in our hearts we know that radiation is not going to "cure" the metastatic lesions. We have recently adopted a more vigorous approach to this situation by use of a combination therapy. The radiologist does not attempt to treat the

neck but focuses his full attention and treatment on the primary lesion. Then, as soon as the course of radiation is completed, a radical neck dissection is performed. Time alone will tell whether this will increase our cure rates, but at least it is a more positive approach, aimed at cure rather than palliation.

Regarding palliative therapy, every case must be individualized. I can only remind you that if a case is obviously and undoubtedly hopeless, it is often better to do nothing than to subject the patient to vigorous radiation or surgical attempts at palliation. On the other hand, we must never hide behind this word "palliation" to excuse ourselves from a vigorous try at curing the patient if it is at all possible.

Having used radiation as our principal form of therapy, we must be ever aware of the "masking effect" of radiation on recurrences. Our follow-up examinations must be especially careful, for a small recurrence can be easily hidden by postradiation edema and tissue changes.

This brings us to the importance of follow-up. These visits must be frequent and business-like. An apparently cured cancer patient is usually a grateful patient, and we must beware of having the follow-up visit become a "social visit."[1] These visits must occur frequently if we are to detect recurrences early.

In summary, may I say that we must avoid at all cost the many pitfalls in the path of early diagnosis and adequate treatment of cancerous lesions of the pharynx. If we don't, no one may ever know—no one but ourselves and our conscience.

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DISCUSSION

DR. MILLER: Before I call on the other members of the panel, I would like to make one or two points.

As far as I am concerned, the palpating finger has a few times discovered pathologic changes which were not even guessed by what was seen.

Another procedure that has helped me to keep from missing a couple of tumors of the nasopharynx is the following: Whenever there is a lesion of the neck proved to be malignant and thought to be of a lymph gland, even though our complete ear, nose and throat examination by every method fails to reveal a primary lesion, we now take blind biopsies of the nasopharynx, especially on the side on which the mass is presenting in the neck. Twice we have been rewarded by making a positive diagnosis, and on one other occasion, blind biopsy of tonsils yielded us a definite diagnosis.

DR. ERICH: I think the most important symptom of malignancy in the oropharynx or the hypopharynx is pain that radiates to the ear when swallowing. Patients, of course, complain of all sorts of symptoms regarding their throats, most of which are functional. But when a patient complains of pain on swallowing, with the pain radiating to the ear, we are immediately suspicious that he has a malignant tumor somewhere in the pharynx and we check very carefully to find it.

There is one kind of tumor on which I feel we should not do a biopsy before treatment is undertaken. We have all seen smooth, rounded tumors deep on the tonsils that may bulge the tonsils into the pharynx a trifle. They can be discovered only on palpation, although some of them are so large that one can see that the tonsil actually is pushed into the pharynx. This smooth tumor is practically always a mixed tumor, a neurofibroma or an adenocarcinoma. If tissue is taken for a biopsy, the capsule is broken and it becomes almost impossible to enucleate it afterwards. You should first ligate the external carotid artery on the affected side, and then attempt to enucleate the tumor through the mouth or externally through the neck. Of course the capsule may be ruptured while the attempt is being made to enucleate

it, but removal of the tissue beforehand only compounds the danger.

DR. MAGIELSKI: I should like to point out that in examination of the pharynx I think we *often* have to discipline ourselves. When we pick up a so-called laryngeal mirror and examine the pharynx, we must also scrutinize the valleculae and the lateral walls of the larynx, and we must look down into the pyriform sinuses.

Secondly, more and more we are now beginning to see people who have been cured of a primary malignant lesion of the pharynx or mouth. This does not mean that the patient cannot develop another lesion in the mouth or pharynx. When we examine him during the follow-up program, we should always make it a point to complete the examination by checking the hypopharynx and larynx carefully.

Symposium: Errors in Early Diagnosis and Treatment of Cancer in the Upper Respiratory Tract

* * *

THE LARYNX

ALDEN H. MILLER, M.D.
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THE larynx, of all the head and neck, is the area in which errors in early diagnosis and treatment of cancer result most tragically in too little therapy being done too late, for many of these cancers could be cured. What are these errors and the safeguards against them?

SYMPTOMS

Hoarseness is the only early symptom of cancer of the intrinsic larynx. We all know that any hoarseness that persists more than two weeks must be proved *not* to be caused by a malignant lesion. Yet, many patients with persistent hoarseness are still being blindly treated with antibiotics and throat medications.

The cardinal symptom of extrinsic carcinoma is *soreness* in the throat, usually one-sided, often aggravated by swallowing, and sometimes accompanied by dysphagia. Failure to appreciate that even a slight persistent sore throat can be caused by an extrinsic malignant lesion has resulted in late diagnosis.

A third complaint is all too frequently overlooked, strange as it may seem; it is that of a "lump, nodule or growth" felt by the patient in his neck. Rarely are these of primary origin. Along with the pharynx and nasopharynx, the larynx must always be suspected and searched.

These three are the important symptoms of carcinoma of the larynx and *all* demand an immediate, completely successful visualization of *all* the hypopharynx and larynx, usually by mirror laryngoscopy but if necessary, by direct laryngoscopy. Failure to do this systematically must be a common error.

FINDINGS

Lesions of the anterior surface of the epiglottis and base of the tongue have been missed because the examiner's attention has gone immediately to the glottis. All walls of the valleculae should be inspected first. The positioning of the epiglottis or the curving of its laryngeal surface has hidden a cancer in its posterior aspect. This must be visualized down to the anterior commissure.

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Failure to see the entire length of the true and false cords, the ventricles and the subglottic area, especially anteriorly, has resulted in missed early diagnoses. Swelling of an arytenoid or of the postcricoid area may hide a lesion further down. But, more important, failure to appreciate that any difference in *size*, or *appearance*, or movement of an arytenoid eminence may represent malignant invasion has delayed a correct diagnosis.

Finally, failure to visualize completely all walls of the pyriform sinuses, especially the posterior surfaces of the arytenoids and cricoid, has resulted in cancer there being missed.

Statistically, cancers of the epiglottis, of the pyriform sinuses, and of the subglottic area are more often overlooked than are those of the cords and aryepiglottic folds.

Only one other procedure is necessary to establish the presence or absence of a malignant lesion: an adequate histopathologic examination of all lesions. Perhaps the greatest error in making an early diagnosis of cancer of the larynx lies in the acceptance of a negative report on a biopsy specimen which the physician suspects of malignancy.

The finding of keratosis only, or of carcinoma in situ, may indicate that the biopsy specimen represented just the fringes of a lesion that is truly neoplastic and invasive. There should be no limit to the number of repeat and deeper biopsies unless the suspected lesion has been completely removed and has rapidly and normally healed.

TREATMENT

The first treatment of cancer of the larynx is the most important, and often it is the only one with a good chance of success. Inadequacy of this initial therapy has been the greatest error. The indications for various operations and for irradiation were beautifully outlined for us by a panel at the Academy in 1953, which consisted of New, Furstenberg, LeJeune, Jackson, Figi, Schall, Orton, del Regato, and Clerf.

The failure to determine the extent of a lesion by meticulous search and multiple biopsies has frequently resulted in the error of planning surgery by laryngofissure for a lesion which cannot be encompassed by this procedure. Failing to recognize the restriction of movement of an arytenoid has resulted in inadequate initial treatment. An operation by laryngofissure is not extensive enough for lesions causing fixation.

Any swelling of an arytenoid usually means that a thyrotomy will not do the job. Any purely subglottic lesion or subglottic extension easily seen usually extends downwards at least a centimeter farther than one thinks it does, and removal requires laryngectomy. Unless the cancer is a purely superficial one, it usually extends much farther intramucosally or submucosally than appearance on the surface would indicate.

The failure to prove microscopically that the lesion removed by laryngofissure has an adequate margin of normal tissue in all directions, has resulted in a laryngectomy being delayed too long.

The desire to preserve the functions of speech and peroral breathing has resulted in the error of choosing roentgentherapy for intrinsic lesions that would have been better treated by surgery. On the basis of my statistics, I still prefer the laryngofissure operation for small, purely cordal cancers, unless the histopathologic examination proves them to be very superficial.

Arytenoid swelling, arytenoid fixation and subglottic extension require laryngectomy, not roentgentherapy, for removal.

Another error that has resulted in the failure of the initial treatment is the failure to appreciate that postirradiation thickening and swelling may hide incomplete regression or early recurrence. The lesion successfully treated by irradiation has usually completely disappeared before the completion of the course, and the larynx has returned to a normal appearance and movement within one to two months after the end of treatment.

Any adenopathy demands radical neck dissection either simultaneously with the laryngectomy or a few weeks later.

Failure to treat carcinoma in situ as if invasion were imminent is an error.

While complete stripping of a cord alone has seemed to give a cure, I prefer a full cancerocidal course of irradiation if there are multiple scattered foci of carcinoma in situ.

More than twenty cases of superficial lesions revealing only carcinoma in situ on repeated examination of biopsy specimens received cancerocidal doses of irradiation. In one patient invasive carcinoma occurred two and a half years later, and three other patients with carcinoma in situ, treated by surgical stripping alone, had invasive carcinoma two years and eight months later, and one patient treated by thyrotomy for carcinoma in situ had a recurrence of it ten months later.

Maybe the recitation of those observed errors seems elementary to most of us, but I believe their implications must at least be preached to our students and our referring colleagues.

DISCUSSION

DR. MAGIELSKI: I should like to repeat the point that many of the patients one sees now with carcinoma of the larynx have received repeated courses of antibiotic therapy on the basis that the patient had laryngitis; many of them have improved temporarily, but in reality the diagnosis has only been delayed.

I think we should also bring home to our colleagues who refer patients to us that one cannot agree to treat a laryngitis without having examined the larynx.

DR. REED: I would like to disagree with you, if I might, Dr. Miller, on the point of advocating the laryngofissure for small, early lesions. I do not see the rationality of this. Recent statistics indicate that the cure rate is just as good with irradiation of the small lesion confined to one freely movable cord as with laryngofissure, and most certainly in the greater percentage of cases the voice is much better following irradiation than it is following laryngofissure. So I see no real reason to advocate laryngofissure for the small lesions except when radiotherapy is unfeasible because of socio-economic factors.

On the other hand, the radiologists like to treat carcinoma of the larynx, and we must be continually alert to keep the radiologists from treating unsuitable cases. Other than in the treatment of these small lesions there is no place for radiation therapy except in treatment of the larger lesions which are beyond the confines of surgery. There has been a tendency, even in recent literature, to say, "Well, we just are not sure yet about the results of x-ray therapy. It has 'cured' some of our patients, so why don't we give the patient a trial of x-ray therapy? If it does not work, we can always do surgery."

Yes, you can always do surgery, but I am sure you are all familiar with the many problems of doing surgery on these patients who have been irradiated previously. We have recently collected a small series of cases and found that the hospital stay of patients who had been irradiated before surgery was from two to three times as long as that of the unradiated patients. That, of course, brings with it a great deal of expense, to say nothing of the blow to the patient's morale when fistulas develop, healing is delayed or poor, and plastic repairs become necessary. So we have to be careful in our initial choice of treatment and avoid the idea of a "trial of x-ray."

If irradiation will do a better job, well, then let's give it credit and use it. If it won't do a better job, then by all means let's use surgery.

HISTORY TAKING IN ALLERGY: USE AND ANALYSIS OF A QUESTIONNAIRE

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INTRODUCTION

SOON after we became interested in otolaryngologic allergy, it became evident to us that it would be desirable to have some type of questionnaire that could be filled out by the patient. A number of standard texts were consulted, but none seemed to provide exactly what we wanted. Nevertheless, several history forms were developed, each being an improvement over the preceding ones. These were employed in our private practice and in the Otolaryngologic Allergy Clinics at the New Orleans Eye, Ear, Nose and Throat Hospital and the Tulane University Medical School.

Eventually we became acquainted with the Cornell Medical Index Health Questionnaire (CMI), developed at Cornell University Medical College under a contract recommended by the National Research Council, between the Veterans Administration and Cornell University. We were impressed by the simplicity of its operation, and it was immediately apparent that a similar history questionnaire was precisely what those physicians who devoted themselves to the diagnosis and treatment of allergic disease sorely needed.

Therefore, a history questionnaire patterned after the CMI was developed, using all the improvements which had been made in the evolution of our previous forms. At first, we gave the patients our questionnaire and, in addition, a CMI to fill out, but then we realized that we needed a form more heavily weighted in the upper respiratory system for the purpose of differential diagnosis. These changes were made.

Finally, we requested permission from Albert J. Erdmann, Jr., M.D., one of the men who developed the CMI, to combine our history form with the Social and Psychiatric portions of the CMI. This permission was kindly granted in the true traditions of Medicine, and the present Allergy History Questionnaire is the result.

We are under no illusions that this is the last word in allergy history taking. We are sure that a better history could be taken if one had the time to spend two or three hours with the patient for that purpose. But we are primarily otolaryngologists who find that cases of allergy comprise a large portion of our practice, and our time is too filled to take such a leisurely history. To us the Allergy History Questionnaire is a time-saving device which permits us to get to the point more quickly.

Finally, we earnestly request that those who use the Allergy History Questionnaire employ it as we recommend it, for our recommendations are based on the successes and failures we encountered in its development.

The object of the allergic history is threefold: (1) to uncover the offending agent or agents, (2) to delineate the pa-

From the Allergy Clinic of the Eye, Ear, Nose and Throat Hospital, New Orleans, Louisiana.
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tient's complete allergic symptomatology, including its development, and (3) to give an insight into all aspects of the patient's medical problem.

Obtaining such a complete history would be too time-consuming for busy otolaryngologists, who, as a group, are notorious for the brevity of their history taking. To meet the need for a method of collecting a large amount of significant allergic and general medical information with the expenditure of a minimum amount of the physician's time, we have developed a questionnaire to be filled out by the patient. Modelled after the Cornell Medical Index Health Questionnaire,[2] the question-and-answer form may be used to uncover promising areas of investigation and thus allow the physician to cut his way directly to the heart of the problem rather than to arrive there by a circuitous route. It also eliminates, to a large extent, the possibility of overlooking what may prove to be important facts in the history.

TECHNIQUE OF USING THE ALLERGY HISTORY QUESTIONNAIRE

The questionnaire consists of 367 questions printed on both sides of four pages. Obviously, this would be too bulky to enclose in a physician's files. To summarize the information obtained and thus conserve space, we have developed a Key, or Summary, Sheet or Card.

If, on the occasion of the first visit, a short history, physical examination and cytologic study of secretions give the physician the impression that the patient's disorder is probably of allergic origin, he is given a questionnaire to fill out. We prefer to have the patient take this home with him rather than complete it in the office, because there it may be done leisurely, usually with the benefit of much clearer thought and also with useful help from other members of the family. When the form has been completed, it is mailed to the office in a self-addressed, stamped envelope provided for that purpose.

It is very tempting to have a secretary transfer the positive information to the Key Sheet, but this practice is mentioned only to be condemned, for it defeats the main purpose of the questionnaire—to give the physician insight into all aspects of the patient's problem. In our hands, less than five minutes are required to study the questionnaire, transfer the pertinent information to the Key Sheet, and establish a provisional program of investigation. As we go through the questionnaire, we place check marks next to the questions we wish to investigate in more detail. On the occasion of the patient's return visit, the indicated areas are thoroughly explored, and any additional information obtained is also recorded on the Key Sheet or Card.

STRUCTURE OF THE ALLERGY HISTORY QUESTIONNAIRE

Aside from questions inquiring about the main complaint and its duration, and about the marital status, occupation and educational attainments of the patient, every question is followed by a "Yes" or "No" to be circled as indicated. As in the Cornell Medical Index Health Questionnaire, the questions are simply worded, medical terminology being avoided for obvious reasons.

The questions are grouped in sections under the following headings:

I. Main Complaint and Its Duration
II. Seasonal Incidence
III. History of First Attack
IV. Discussion of Attacks
V. General Allergic Symptomatology
   A. Nose
   B. Throat
   C. Ears

D. Eyes
E. Chest
F. Gastrointestinal System
G. Skin
H. Miscellaneous

VI. Childhood Allergic History
VII. Family History
VIII. Nonpollen Inhalant History
IX. Food History
X. Physical History (Trigger Mechanisms)
XI. Focal Infection History
XII. Environmental Survey
XIII. Previous Treatment History
XIV. General Medical History
XV. Social History
XVI. Psychiatric History

We will now discuss the various sections in detail, indicating what information is being sought. For the beginner in allergy, this might be termed a lesson in how to "think allergically."

Address of the Patient

Though seemingly too trivial a thing to consider, the address of the patient may give some helpful information.

Knowing the general locality in which he practices, the physician may correlate the location of the patient's home with factories in the vicinity, certain low, damp areas which might be the source of luxuriant mold growth, or wooded areas or fields which might be the source of pollen, to name but a few possible influences. If the address is in an old section of town, the dwellings may be either damp or extremely dusty. A rural address may direct attention to allergens commonly found in those locations or around farms, e.g., insect and vegetable sprays, animal danders, rusts, and smuts. Then, also, every physician treating allergic patients should have some basic knowledge of micrometeorology so that he might assess its influence when a patient lives in a certain locality.

Occupation and Duration of Occupation

Every individual is exposed to allergens or irritants peculiar to his particular type of work. A relationship between the onset or exacerbation of symptoms and the beginning of employment may exist; or, perhaps, symptoms may be dated to the institution of some new process in the course of work. Standard texts contain listings of the chief allergens encountered in various occupations.

Main Complaint and Its Duration

The patient often makes his own diagnosis because his symptoms are so obviously of an allergic nature; thus, he may list his main complaint as "hay fever" or "asthma." At times he will be told by some other physician that he is suffering from an allergic disease and so will list it simply as "allergy." In most instances, however, patients will list one or more symptoms which bother them most, overlooking numerous others which are not so annoying.

The duration of the main complaint does not necessarily signify that allergic symptoms began at that time, for at this point in the allergic investigation, patients have a tendency to think only in terms of major attacks, whereas they may have actually been experiencing minor symptoms for a long time prior to this.

Correlation of the duration of the chief complaint with the age of the patient may give valuable information. Food sensitivities are very frequent in infants and young children because of the immaturity of their digestive tracts and the fact that massive exposure is likely to occur due to the limited nature of the diet. Bacterial allergy also seems to be uncommonly frequent in children; this is explained, in part, by the increased incidence of respiratory disease and the poor drainage of secretions from

the upper respiratory tract in children. Many allergic conditions make their first appearance at puberty or around the time of the menopause and appear to be related to the great hormonal and emotional changes which occur during those periods. When allergic complaints begin late in life, bacterial allergy should be strongly suspected, and a thorough search for foci of infection should be conducted.

As we have indicated previously, there may be some relationship between the initial development or exacerbation of symptoms and the duration and type of the patient's employment.

The time of onset of symptoms may also be compared with the date of the patient's marriage, separation or divorce, the birth of a child, or the death of a mate, for these are times of great emotional upheaval, and any such relationship should prompt one to be on the lookout for a large psychosomatic element in the case.

Seasonal Incidence (*Questions* 1 *through* 4)

Seasonal incidence or exacerbation of symptoms is investigated mainly to determine whether pollen allergy should be suspected. However, it will also give other information such as the relationship of symptoms to certain seasonal foods, activities, or types of living.

Limitation of symptoms to the spring of the year should cause one to investigate the possibility of tree pollen sensitivity. It is not uncommon for springtime exacerbation of symptoms to be due to massive exposure to house dust or paint fumes during spring house cleaning and painting, contactants in gardens, and seasonal foods.

Grass, chenopod, amaranth, dock, plantain and thistle pollen should be suspected, depending on the locality, when the disease is limited to the summer months. Atmospheric mold is also most prevalent at this time of the year, as are a number of allergenic insects. Contactants in gardens, fields and sports should also be investigated. Again, the possibility that some seasonal food is the offender should be considered.

The main suspects in the fall are ragweed, amaranth, sage, thistle, and kochia pollen, depending on the area. In a few states, grass and tree pollen may be sufficiently plentiful because of the temperate climate, to precipitate symptoms. Damaging concentrations of atmospheric mold, rusts and smut may continue on into the fall. House dust assumes increasing importance at this time because householders begin to keep windows shut for longer periods, central heating systems are turned on, rugs are taken out of storage for use, and heavy blankets and clothing containing dust are again put into use.

During the winter, the main suspect is house dust, but tree pollen and atmospheric mold may play a part where the year-round climate is mild. Seasonal foods may also be important. Bacterial allergy should be considered because of increased incidence of respiratory infection due, in part, to the frequent temperature changes to which the individual is exposed.

Should the answers indicate that symptoms are present perennially, i.e., during the entire year, the offending allergens are probably substances to which the patient is constantly exposed.

History of First Attack (*Questions* 5 *through* 7)

Gradual onset indicates a slowly progressive development of sensitivity, usually to a common environmental allergen or group of allergens. Such a history should also cause one to think along

quantitative lines, for it has often been said that a person might become allergic to anything provided the exposure is great enough and sufficiently prolonged.

Acute onset points either to the introduction of a new antigen into the environment (such as a new or unusual food; a change in residence, occupation, furniture, rugs, or clothing; or new household pets), massive exposure, or some unusual occurrence which lowers the defensive mechanism of the body (such as a sinus or upper respiratory infection, a gastrointestinal upset, or such acute infectious diseases as measles, whooping cough, and diphtheria).

At times the patient may be helpful in bringing out some fact that the investigator might overlook; therefore, it should be routine to ask the patient if he has any idea what caused the condition to begin.

Discussion of Attacks (Questions 8 through 15)

In this section of the history, an attempt is made to obtain information about the individual attack, in contradistinction to the last set of questions, whose purpose it was to clarify the circumstances surrounding the onset of the disease itself.

As previously stated, gradual onset indicates a slowly progressive development of hypersensitivity to a common environmental allergen or group of allergens. In either case, the quantitative aspect of the problem should be considered.

Acute onset might indicate the introduction of a new antigen into the environment, massive exposure, or the presence of some unusual condition which lowers the threshold of allergic symptoms.

Inquiry concerning the frequency of attacks is directed toward establishing, if possible, the frequency of contact with the allergen or the frequency with which the allergic load[1] becomes great enough to produce symptoms.

The duration of the attack sometimes indicates the degree of sensitivity of the patient or the duration of exposure to the offending allergen.

Attacks occurring after the patient has retired should direct one's attention to some article of bedding or something in the bedroom. The widespread use of window fans and attic ventilation during the summer months has created a specialized set of problems inasmuch as abnormal amounts of pollen and other irritants are pulled into the bedroom and aggravate the allergic individual, who often sleeps directly in the path of air flow; in addition, when the patient turns about in bed, warm parts of his body are chilled in the draft, which then acts as a physical allergen or serves to trigger the allergic reaction. Rinkel[4] reports that attacks occurring in the middle of the night often indicate food sensitivity.

At times, measures which the patient states have given relief in the past may help not only to establish the disease as allergic in nature but also to give a clue as to its etiology. There are numerous examples. Relief from antihistaminic drugs is the outstanding one. If improvement is reported following the use of vasodilator drugs, a physical allergy may be suspected. Lessening of symptoms after a course of antibiotic therapy may occur in bacterial allergy. Relief of hay fever symptoms as a result of a trip to other areas of the country may occur in cases of pollenosis.

It is desirable to establish the severity of the condition. A mild allergy may not warrant an extensive diagnostic and therapeutic program; for these individuals it may be economically more prac-

tical to take care of their acute episodes symptomatically. The same may be said for the patient who has an acute episode for a short time once a year, as in the two-week tree-pollinating period, for example. Should the attacks be severe, it will be necessary to give the patient symptomatic treatment in addition to elimination and hyposensitization.

Nasal Symptoms (Questions 16 through 28)

It is indeed rare to find a person who presents but one manifestation of allergy; usually, however, patients do not realize that many of their minor complaints are but part of an over-all picture. A complete knowledge of the individual's allergic symptomatology is valuable to the physician for several reasons. First, the location of the shock organ may give clues as to the types of allergens involved; e.g., in the respiratory system, our main interest, inhalant allergens are the most common offenders, with bacteria and foods in an important, but secondary, role. Secondly, if symptomatic relief is indicated, it may be more completely achieved. Finally, it may be of value in following the response to therapy and in the evaluation of the role played by infection, anatomic derangement and psychosomatic influences.

Sneezing frequently follows itching in the nose and these two are among the most prominent symptoms in cases due to inhalant allergens. They may result from actual contact with the allergens involved, but apparently, almost as frequently they occur because the allergic nose, being hypersensitive to sensory as well as allergenic stimulation, is "triggered" by such physical irritants as heat, cold, fumes, odors, sunlight, dusts, and slight trauma (with a handkerchief, for example). A typical manifestation of the "trigger" phenomenon is the sneezing attack which allergic patients often experience in the morning upon arising. This is felt to be due to the chilling produced when they emerge from under the bed covering and their bare feet first touch the cold floor, although undoubtedly circulatory readjustment may play a part.

Blockage of the nasal air passages in cases of allergy is of course due to tissue edema and varies in degree, duration and laterality depending on severity of hypersensitiveness, amount of secondary tissue change (hyperplastic fibrosis) and disturbances in the anatomical configuration of the internal and external nose.

Before leaving this subject, it is felt that a short discussion of a very important differential diagnostic point is in order. Patients often speak of the nose being blocked when the word "stuffy" would more accurately describe the condition. Apparently, to experience a sense of normal breathing, a certain amount of negative pressure must be developed on inspiration and a definite degree of moistening of the inspired air must occur. Extremes at either end of the scale will result in a sensation of "stuffiness" which the patients loosely describe as "blockage." Examples of this are common clinically; patients with markedly deviated nasal septums often complain of blockage on the side which is most patent, and those with atrophic rhinitis without crusting report that they suffer from nasal blockage even though there is actually an increase in the volume of the internal nose.

Aside from allergy and infection, the most common causes of nasal stuffiness in our experience have been reaction from fear and anxiety, excessive smoking, excessive coffee drinking, and endocrine hypofunction due to advancing years.[1a] Fear and anxiety cause stimulation of the sympathetic nervous system

and consequent release of an epinephrine-like substance called sympathin. Tobacco smoke, in addition to being a local irritant, acts systemically and also produces vasoconstriction. Coffee, as a result of its caffeine content, also causes vasoconstriction and, because it is a potent diuretic, causes dehydration and thickening of the nasal mucus. Anything that causes vasoconstriction in the nasal mucous membrane will cause a decrease in the amount and an increase in the viscosity of the mucus produced. Thus, the nasal passages may be open widely, yet the patient will say that they are blocked. Usually these patients complain of a seeming paradox: they report a great deal of dryness of the nose and throat, yet complain bitterly of the thickness of the mucus and the difficulty they experience in trying to rid themselves of it.

This latter syndrome (dryness and burning of the membranes of the nose and throat, stuffiness of the nose and the presence of thick, tenacious mucus which is difficult to expel) might be termed the upper respiratory sympathetic syndrome. It should not be confused with allergy and may prove to be a pitfall for the unwary. It may occur in patients with latent allergy or as an independent entity; in the latter instance it may be mistaken for allergy. Prescribing antihistaminic drugs in these cases will serve to aggravate them. Nasal smears with Hansel's stain will reveal the presence of a large amount of thick mucus with only a few scattered cells which are not eosinophils. Finally, the allergic patient may give a history of having to clear a large amount of mucus from the nasopharynx, but it is never as viscid as in the sympathetic syndrome unless there is a great deal of anatomic obstruction in the nose; in the latter instance there are always diagnostic amounts of eosinophils in spite of the thickened mucus.

Headache associated with nasal blockage is not infrequent among allergic individuals. The nasal blockage may prove to be the direct cause of the headache, or it may be just one incidental manifestation of the circulatory dysfunction causing the pain. Nasal mucosal swelling may be directly causing the headache because of the establishment of pressure points between the turbinates and the septum, between adjacent turbinates, or between the turbinates and the lateral wall of the nose. The individuals so constituted also experience headache when the nasal mucosa swells from other types of irritation. In these cases a submucous resection may have to be considered. The diagnosis of this mechanism of headache production is easily made by determining the effect of selective field-block anesthesia while the patient has a headache.

A prime manifestation of allergy is increased glandular secretion. The mucus produced may vary in amount, in consistency, and in the way in which it bothers the patient. At times it is very watery, as in hay fever, and so profuse that it will drain from both the anterior and posterior nares. Generally it is more mucoid and is cleared from the nose either by blowing or by the cilia, which carry it posteriorly into the nasopharynx. The most viscid mucus is found in those cases with marked obstruction of the nasal passage (e.g., marked deviation or spurring of the nasal septum, and nasal polyposis); in such instances the viscid mucus will accumulate in large quantities in the nose. A particular complaint of allergic patients concerns the large amount of mucus which they have to clear from the nasopharynx each morning upon arising. This represents a pooled accumulation over the sleeping hours. At times it becomes particularly aggravating because of the increased viscosity due to mouth breathing.

As offensive as it may be to his sensibilities, the allergic patient will usually find that the mucoid postnasal discharge does not have an unpleasant taste. If there is superimposed infection, particularly when a sinus is involved, the discharge changes color and has a sweetish taste which is nauseating and particularly disgusting.

An overwhelming percentage of nasal polyps are of allergic origin and will recur following removal if the patient's allergy is not controlled.

Many individuals with nasal allergy have been under the impression for many years that they are suffering from "sinus trouble," the implication being that the cause of their difficulty is some chronic and incurable infection. This is caused by the vague conception of sinusitis in the mind of the public and also by the fact that many physicians who are not trained in allergic diagnosis and therapy treat allergic disorders as though they were infections. Strangely enough, this latter state of affairs may go on for years, for there are certain phenomena associated with allergic disease which work on the side of these physicians, the main one being the fluctuation in severity of allergic symptoms.

The patient is asked whether or not he has hay fever because, being preoccupied with other symptoms, he may overlook the seemingly obvious. The presence of seasonal hay fever usually implicates a seasonally prevalent pollen.

Anosmia frequently accompanies the more severe types of nasal allergy. It may be due to the fact that nasal obstruction prevents odors from reaching the olfactory nerve endings in the roof of the nose. Sometimes it will disappear during periods of quiescence or when the allergy is controlled; in other instances it is permanent, perhaps because of metaplasia of the epithelium in the olfactory sulcus because of the presence of nasal polyps.

Allergic patients quickly learn to differentiate the onset of an upper respiratory infection from the onset of an allergic crisis. If an allergic patient has frequent colds, or if his cold seems to hang on for a long time, one should suspect the possibility of bacterial sensitivity or at least the presence of a localized loss of immunity to the bacteria concerned. Of course this does not mean that a bacterial allergy may not be operative in the absence of affirmative answers to these questions. In fact, it is felt that bacterial sensitivity should be considered whenever there is chronic blockage of the normal flow of secretions, whether it is due to chronic vascular engorgement of the nasal structures or to anatomical obstruction in the nose or nasopharynx.

We are aware that the question of bacterial sensitivity is a controversial one among allergists. Yet proof of the pudding is in the eating, and our beliefs are based on the good results often obtained with bacterial vaccine therapy in patients who seem to have frequent or prolonged nasal infections.

Throat Symptoms (Questions 29 through 37)

As was mentioned previously, itching is one of the most prominent symptoms of allergy.

Individuals with nasal allergy often complain of sore throat being present in the morning when they awaken but disappearing after they have been up and about for a while. Because of the nasal blockage, these individuals sleep with their mouths open, often snoring, and the unwarmed inspired air dries out the pharyngeal mucosa and gives rise to pain. When they awaken in the morning, this condition is reversed by several factors: when the upright position is assumed, the nasopharynx is cleared of accumulated secretions and nasal breath-

ing is resumed; breakfast and hot coffee, or some other beverage, stimulate the production of mucus and sooth the membranes, and the mucus produced again follows its normal pathway from the nasopharynx, down the pharynx, to be swallowed.

Clearing the nasopharynx of the secretions which have accumulated overnight is sometimes so difficult that violent effort is required. This, in turn, may cause nausea, and some individuals actually vomit.

Postnasal discharge, hypopharyngeal accumulation of mucus and, undoubtedly secretions carried upward in the trachea by ciliary action, often cause the allergic individual to want to clear something from between his vocal cords. Should his efforts be violent enough or persist over a long period of time, vocal cord edema with subsequent hoarseness or huskiness of the voice often ensues.

Ulcerative stomatitis, glossodynia, excessive redness of the tongue, and swelling of the lips and tongue, as well as the salivary glands, are often due to allergy. They may result from sensitivity to foods, drugs, or anything that passes the lips, e.g., tobacco products, lipstick, dental plates, tooth pastes or powders, mouth washes and chewing gum.

Ear Symptoms (*Questions* 38 *through* 44)

The possibility that pruritis of the external ear canal may be of allergic origin should be kept in mind. In most instances, however, this is not the case.

Intermittent blockage of the eustachian tubes as a result of allergic edema is often the cause of recurrent ear pain without signs of local inflammation. This is often associated with the development of secretory otitis, in which case the patient will frequently volunteer the information that he experiences the sensation of a drop of water in the ear. As a result of these changes there is interference with the normal function of the ear, and hearing loss, tinnitus and vertigo occur either singly or in various combinations. However, it is well to remember that allergy may be the cause of certain vasomotor phenomena of the inner ear which result in the production of these same symptoms. In either case the outstanding characteristic is the fluctuating nature of the symptoms.

Many cases of chronic drainage from the middle ear are perpetuated by allergy, and eosinophils in diagnostically significant numbers may be found in the mucoid material obtained from the ear. It is impossible to obtain a dry ear in these cases unless the allergic element is controlled.

Eye Symptoms (*Questions* 45 *through* 53)

The questions in this section are designed to elicit some of the frequent complaints of allergic individuals even though the tissues of the eyes are not the main shock tissues.

The most commonly encountered manifestations of allergy of the eye are involvements of the conjunctiva and the periorbital skin; however, numerous reports in the literature indicate that every portion of the eye can be affected by allergy.

As Hansel[3] points out, the conjunctival epithelium morphologically lies somewhere between the ciliated epithelium of the nose and that of the skin, so allergens affecting those two structures may be factors in the production of conjunctivitis and blepharitis.

Among the reported causes of allergic dermatitis of the eyelids are dyes for hair, eyebrows or eyelashes; shampoos; foundation creams; cosmetics; nail polishes; drugs; insecticides; oils

of plants; spectacle frames; rubber; dandruff; paint; wave-set lotions, and pathogenic fungi.

Chest Symptoms (Questions 54 through 56)

Cough is the most frequent respiratory manifestation of allergy, yet the allergic etiology is frequently unrecognized unless it is associated with bronchial asthma. In the absence of any demonstrable cause, a cough which has not responded to the usual sedative cough mixtures should be suspected of being due to allergy. Several types of allergic cough are recognized.

There is a hacking, nonproductive cough which may occur at any time during the day or night. It resembles smoker's cough and may be mistaken for it. In children, however, it characteristically begins soon after they retire and may continue periodically through the night, causing a restless sleep. Sometimes patients will report that it is accompanied by an itching sensation in the throat or chest.

Another type of cough due to allergy is associated with mild nasal stuffiness, with postnasal discharge, and often with frequent attempts at "clearing" the throat.

A third type is that accompanying so-called "allergic" or "asthmatic" bronchitis. Here there is mucus in the respiratory tree but the lumen is not occluded enough to produce the typical wheezing of asthma. Patients often say that their attacks of asthma begin suddenly, but upon careful questioning they will admit that they have had cough and what they describe as "congestion" in the chest for some time. It should be pointed out here that the condition is often associated with purulent sinusitis and will subside when the sinuses have received proper treatment.

Finally, there is the cough associated with bronchiectasis complicating allergic respiratory disease. In these instances, the cough will be productive of more purulent and foul-smelling secretion than is generally seen in uncomplicated cases.

As implied above, coughs due to allergy do not respond to the usual sedative cough mixtures but will frequently show quick improvement when the patient is given ephedrine or some other bronchodilator. Good response is sometimes obtained with antihistaminic drugs when the cough is a dry one.

Should the patient state that he is bothered with coughing, the physician should explore the area thereby opened. He should try to determine, among other things, whether the cough is productive or nonproductive, the characteristics of the mucus if the cough is productive, whether it is present only with colds, whether it is associated with elevations in temperature, whether it is paroxysmal and/or seasonal, and if there is an association with exertion.

Many patients do not know what asthma is, but this is the simplest way of asking whether the individual is suffering from recurrent paroxysmal dyspnea associated with wheezing and cough. What the physician should keep in mind is Chevalier Jackson's time-worn and honored dictum, "All is not asthma that wheezes." All standard texts contain lengthy lists of conditions which may cause dyspnea and which may be mistaken for asthma. Many texts have tables differentiating bronchial asthma from cardiac asthma. A positive answer to this question should indicate further investigation by the physician upon the occasion of his second interview with the patient.

Many individuals seem to have repeated attacks of bronchial infection as-

sociated with the asthmatic syndrome. Cough, with or without low-grade temperature, characterizes the condition. The most frequent cause of these attacks is a bacterial allergy, and a focus of infection is very often found in the paranasal sinuses.

Gastrointestinal System (Questions 57 through 68)

All of the symptoms in this section have been reported as being due to food allergy, and their presence in a case of otolaryngologic allergy should cause one to consider the possibility of food sensitization. Foods usually are not primary allergens in these cases but act by increasing the allergic load; however, it should be remembered that foods play a more important part in the production of symptoms in children than in adults.

Skin Symptoms (Questions 69 through 77)

The most commonly encountered dermatologic manifestations of allergy are contact allergic dermatitis, atopic or familial allergic dermatitis (includes atopic or infantile eczema and generalized atopic dermatitis of older children and adults), urticaria and angioedema. The type depends on the particular portion of the skin in which the allergic reaction primarily occurs.

The otolaryngologist most frequently deals with allergic contact dermatitis in the area of the external ear. Some of the reported causes of this condition are ear muffs, earrings, goggles, spectacle frames, hearing-aid ear inserts, hair nets, medications, perfumes, pillows, hair rinses and shampoos, scalp lotion, shower caps, telephone receivers, anything used for scratching the ear, and anything brought into contact with the ear by the hands.

Urticaria and angioedema are but different phases of the same process: in urticaria the site of reaction is the vascular bed of the corium, but the deeper vessels are involved in angioedema. The otolaryngologist may see these lesions about the mouth, the lips, the tongue, and the mucous membranes of the mouth, uvula, pharynx and larynx. It is generally believed that most cases of this type are due to food allergy, but they may also be caused by ingested or injected drugs and inhalants. The allergens are carried to the site of the lesions by the blood stream.

Miscellaneous Symptoms (Questions 78 through 84)

We have already considered mucosal edema of the nasal passages as a cause of headache.

A fairly common type of headache is due to myalgia of the muscles of the head and is said to be due to an allergic reaction within the muscle itself. It is a recurrent condition and is sometimes characterized by the presence of firm, tender areas in the muscle; the pain is disturbed along myotomic segments and it is of a deep, aching type which gradually increases in severity and gradually subsides. Response to vasodilator drugs is good. Food allergy, physical allergy, or psychosomatic stimuli may precipitate the attacks.

There has been much discussion as to whether migraine and migraine equivalents are of allergic origin. Undoubtedly they are in some instances due to food allergy.

Unusual sleepiness, inability to think correctly, and difficulty in concentration are not infrequently seen in patients with the allergic syndrome; they should make one think of the possibility of cerebral angioedema. Food allergy has been reported as the cause of some cases of epileptiform convulsions. The same is true, to some extent, of excessive tiredness.

Many patients designate as "rheumatism" any recurring, aching pain occurring in the region of the joints or in muscle. There is some evidence that these are of allergic origin in some cases at least. They resemble allergic myalgia of the head.

The familiar serum sickness which follows injection of foreign serum is, of course, an allergic manifestation.

The last question of the section lists a number of commonly used drugs which have been found to cause allergic symptoms. Among the symptoms encountered are increased temperature, various types of skin eruptions, itching, edema, swelling of the lymph nodes and joints, leukopenia, granulocytopenia and purpura.

Childhood Allergic History (*Questions* 85 *through* 98)

It is well known that untreated allergic patients often demonstrate a progression of syndromes during their lifetime. Thus, it is not unusual to discover that the patient who reports that his trouble began only recently, in reality had been having allergic symptoms of various types since childhood. Children with allergic (atopic, infantile) eczema usually develop some other syndrome, such as perennial allergic rhinitis, as they grow older. Again, many people with uncontrolled hay fever later develop asthma.

In addition to its value as confirmatory evidence of allergic etiology, this information is of value from another point of view. Very often the physician discovers complications of the allergic state when he is first consulted by the patient. Knowledge of the allergic manifestations of childhood will allow him to evaluate the complication more adequately and thus aid him in estimating the probable efficacy of therapy and the prognosis of the case.

Family History (*Questions* 99 *through* 110)

Information obtained in the family history is of value in several ways. It is confirmatory and aids in the establishment of a probable allergic etiology. When the clinical history is uncertain, a positive family history causes one to give more consideration to a possible allergic cause, for the greater the indidence of allergic disease in one's antecedents, the greater is the likelihood that one will develop an allergy.

Nonpollen Inhalant History

House Dust (*Questions* 111 *through* 116)

House dust is the most frequent cause of respiratory allergy; therefore, certain characteristics of house-dust sensitivity are worthy of note.

Because house dust is everywhere, symptoms may be present throughout the year, with exacerbations occurring because of the individual's particular living conditions and activities. Generally speaking, symptoms are worse at home—more particularly in the bedroom; therefore, they seem worse when the patient is confined indoors, such as on rainy or cold days. Symptoms are also worse during the winter when the windows are kept closed and the humidity of the air in the house is decreased. Exacerbations usually occur as a result of massive exposure during such house cleaning activities as sweeping; making beds; fluffing pillows; turning mattresses; cleaning picture frames, woodwork, radiators, heaters, and closets; beating rugs; cleaning overstuffed furniture; using old-style vacuum cleaners; and moving or cleaning window shades, venetian blinds or draperies. Flare-ups are often noted when the first cold snap of fall arrives and the heat is turned on in central heating systems or floor furnaces, when dusty books or magazines

are handled, and when visits are made to such dusty places as theaters, churches, libraries, grocery stores, schools and department stores. Symptoms seem to improve when the patient is away from the house and during the summer when the windows in the house are left open and there is more outdoor living.

Atmospheric Molds (*Questions* 117 *through* 123)

Next to house dust, we have found that mold spores are the most common cause of nonpollen respiratory allergy. Mold allergy may stimulate pollen allergy, or the two may be [illegible].

Patients with this type of sensitivity usually have symptoms coincidental with elevations of the mold spore count such as occur during or following periods of high humidity. Since molds are plant saprophytes and live mostly on dead and injured vegetation, mold-sensitive individuals may report aggravation when they are around freshly cut grass, cereal grain, hay or weeds, when going to circuses, when entering barns or when being nearby at the time animals are being fed. A particularly abundant growth of molds is frequently found in old, damp houses, basements, sheds or cellars. Quite frequently, especially in humid climates, there is a luxuriant growth of mold on stored luggage, shoes, gloves, canvas, and bread or other exposed foods; thus, patients may recall that symptoms occur or are aggravated when they enter lockers or other storage areas or when they eat such foods as cheese, beer, mushrooms, aged meat, bakery products containing large amounts of yeast, cured hams, and certain citrus fruits. There is also a high mold content in old overstuffed furniture or mattresses.

It should be noted that micrometeorology of the area in which the patient works or lives should be taken into consideration. Thus, mold exposure may be greater among those who live in valleys, near swamps or near grain fields.

Even a patient's hobby may be a source of contact. Gardeners, equestrians, hunters, livestock breeders and even fishermen are exposed to high concentrations of mold spores.

Feathers (*Questions* 124 *through* 128)

Feathers are most commonly contacted via pillows, upholstery, mattresses, down comforts, articles of clothing, and pets. Chicken, duck, goose, pigeon, parrot, swan, canary, turkey and sparrow feathers are the most frequent offenders.

Feathers may become occupational allergens among the following workers: those engaged in mattress, pillow and furniture manufacturing or sales; farmers; artificial flower producers; seamstresses; hat makers; those working in pet shops; and those who raise, ship or market poultry.

Paper (*Questions* 129 *through* 131)

Paper sensitivity is not too uncommon in our experience. Small particles of paper become detached from the roughened edges of newspapers and books. Another source is the commonly used cleansing tissue, such as Kleenex; when these tissues are carried around in one's pocket or purse, small particles are apt to become detached and act as inhalant allergens.

Persons who work as shipping clerks or wrappers in stores may develop symptoms from the inhalation of paper.

Smoke (*Questions* 132 *through* 134)

As was mentioned previously, allergic mucous membranes are hypersensitive to sensory as well as allergenic stimulation. Therefore, tobacco smoke may act as a simple irritant, and most

allergic patients will report that they are adversely affected by tobacco smoke. On the other hand, the response of the nasal mucosa may be a truly allergic one.

Orris Root (*Questions* 135 *through* 137)

Orris root is a fine powder, derived from the iris. It has a tendency to bind odors. It, therefore, has been used as a base for powders, perfumes and other cosmetics. Publicity given to the fact that it may be a potent allergen has caused most cosmetic manufacturers to abandon its use, although it is still used in the cheaper cosmetics.

Possible sources of orris-root contact are face powder; talcum, dusting and tooth powders; shampoos; scented soaps; perfumes; rouges; lipsticks; cosmetic and shaving creams; lotions and sachets.

Exposure is frequent in places where people gather, such as parties, theaters, and churches, and in beauty parlors and barber shops.

Insecticides (*Questions* 138 *through* 140)

Insect powders, sprays and repellents may act either as primary irritants or as allergens. The most common offenders in these products are pyrethrum, derris root, lethane and paradichlorobenzene.

Cottonseed-Flaxseed (*Questions* 141 *through* 142)

Cottonseed may be an inhalant allergen either in the form of the cotton linters, which contain fragments of the seed, or in the form of cottonseed meal products. Cotton linters are contained in cotton wadding or batting used to make mattresses, pillows, pads, cushions and comforts. Cottonseed meal products include fertilizer, food for cows, horses and other animals, and such foods for humans as flour, gin, cooking oil and oleomargarine. Cottonseed meal is also used in certain cosmetics.

Flaxseed as an inhalant is not a very common cause of respiratory symptoms. The main sources are cattle and poultry feeds, such products used in beauty and barber shops as wave-sets, shampoos, and tonics, and finally, some paints and varnishes.

Animal Hairs and Danders (*Questions* 143 *through* 144)

These substances may be contacted either in the living animal or in materials made from hair or hides.

Food History (*Questions* 145 *through* 151)

Two types of reactions to food allergens are recognized.[4] The first is the so-called fixed or permanent type; a food causing this type of allergic reaction will precipitate definite and often severe symptoms whenever it is eaten. This type is relatively easy to diagnose by history, and the patient will usually quickly volunteer the information desired, often without solicitation. The second type is the cyclic form; a food causing this type of allergy will become a more potent allergen the more it is used, the individual tending to develop tolerance when it is omitted from the diet for a period of time. Diagnosis of this type of sensitization is often difficult.

With the above facts in mind, one should determine whether or not the patient suspects that certain foods cause or aggravate his condition. If he does suspect a certain food, inquiry should be made as to whether biologically related foods also cause symptoms.

Food dislikes and disagreement are sometimes indicative of a food allergy. However, it should be remembered that these may be due to fads, eating habits,

faulty training in childhood, whim, or certain psychic influences. Organic gastrointestinal disease may also be at fault in these cases.

Overeating, or the ingestion of large amounts of a given food day after day or at frequent intervals, tends towards producing sensitization. This may result from a particular fondness for one type of food, the seasonal availability of the food, dietary habits, or economic or social status (elderly people who live alone are particularly noted for the monotony of their diets). At times, people who are on one type of diet or other may become sensitive to a food contained therein because of the limitation in variety and the repeated ingestion of the particular food.

The most frequent offenders among foods are wheat, eggs, milk, corn, potatoes, oranges, peas, beef, tomatoes, coffee, lettuce and pork. It is wise for the physician to inquire about the foods individually to see whether there is frequent ingestion, whether they produce symptoms, or whether the patient avoids them for any special reason.

Physical History (*Questions* 152 *through* 166)

Physical agents may affect the allergic individual in two ways: (1) they may aggravate or ameliorate the symptoms of ordinary pollen, inhalant, food, bacterial or contact allergies (in this manner they may be said to be acting as "triggers"), or (2) they may produce symptoms in the absence of the above sensitivities, and these symptoms can be relieved by an application which is the reverse of the one which caused the reaction.

The factors in the first category are, in reality, contributing factors which are incapable of producing an allergic attack themselves, but they adversely affect capillary function in the same direction as does the allergic reaction. Thus there is an increase in the allergic load and a production of symptoms. The proof of the last statement is frequently demonstrated clinically when patients report that such factors as weather change will precipitate asthma at times but not at others.

Those factors in the second category (light, heat, cold, mechanical irritants) are exciting factors and resemble, if they are not identical to, true allergens. In some instances this can be proved by passive transfer.

Focal Infection History
(*Questions* 167 *through* 180)

There is a great deal of controversy among allergists as to whether or not bacterial sensitivity exists. For reasons previously presented, we believe that it does; therefore we feel that a search for focal infection is indicated.

The patient is questioned concerning possible foci in various locations. The most important foci in respiratory allergy are those found in the paranasal sinuses, in the tonsils and adenoids, and in bronchiectatic dilatations in the lungs. It is not sufficient to eliminate these foci mechanically; autogenous vaccines should be made for a program of hyposensitization.

Environmental Survey
(*Questions* 181 *through* 219)

Old homes are more likely to have great amounts of house dust; new ones often do not dry out for several years, so the growth of mold may be favored and humidity control may be a problem. Nearby factories or railroads may be the source of noxious smokes. If a barn is in the vicinity of the patient's home, one should consider the possibility that certain animal danders are among the offending agents. Feathers should be suspected if poultry is kept on the premises. Swampy areas are potent sources

of mold spores. Pollen from nearby lots overgrown with weeds or trees may be important allergens. Fumes from open gas heaters and smoke from open fireplaces may be irritating to the allergic nasal mucosa. The circulation of dust should be kept in mind when heating is achieved with hard-to-clean radiators, central systems with ducts, and floor gas furnaces.

As mentioned previously, window fans and attic fans are often injurious to allergic individuals because they pull large amounts of pollen into the house; window (and to a lesser extent, central) air-conditioning may aggravate an allergy which is triggered by chilling.

Accumulations of books and magazines, pictures on the wall, overstuffed furniture, rugs, rug padding, draperies, stuffed birds and animals, and trophies are all possible sources of dust in the home.

Pets in the house or the yard may be the source of allergenic animal danders. Pillows and down comforts may be the source of feathers. Kapok is often found in small throw pillows. Books, furniture and wallpaper are sources of glue. Paints, linoleum and oilcloth contain cottonseed and linseed oil. Insecticides and mothproofing materials contain many irritating substances, the most important one being pyrethrum.

Previous Treatment History
(*Questions* 220 *through* 224)

A history of previous therapy may be helpful for a number of reasons. Good response to antiallergic therapy tends to confirm the allergic etiology of the complaints; good response to some antiallergic medications and poor response to others will allow the physician to eliminate the ineffective ones from his program immediately with a resultant large saving of time and money. Only scratch tests may have been done in previous investigations; it is well known that many allergens undiscovered in scratch testing will give positive intradermal tests. Severe reactions during testing and treatment may be avoided at times. Previous surgery may have adversely affected upper respiratory physiology to such an extent that the improvement, even with the most skillful therapy, will be disappointing. In some cases, the patient may still be taking previously prescribed medication which would interfere with various diagnostic measures. Finally, one is able to determine whether or not previous diagnostic and therapeutic measures were adequate not only in regard to type but also as to duration.

General Medical History
(*Questions* 225 *through* 308)

The importance of obtaining a general medical history during every allergic investigation cannot be overemphasized. Allergic management has so many inherent pitfalls for the unwary physician that he should not ignore the field of differential diagnosis. The patient may be suffering with a condition of much more serious import than the one for which the allergist is consulted: bronchogenic carcinoma may cause wheezing and dyspnea even in the allergic patient; some systemic disease may have to be cared for before proper allergic control can be attained; anemia, malnutrition, hypothyroidism and other endocrine dysfunctions are but a few of the conditions frequently encountered. Then, also, the presence of an associated systemic condition may make the use of certain antiallergic drugs hazardous: the use of epinephrine or ephedrine in hypertension, and the use of cortisone in persons with a past history of tuberculosis are cases in point. A wealth of information useful in the diagnosis and management of the allergic patient may be obtained from the medical history.

Social History (Questions 309 through 316)

The social history often gives the physician some insight as to the type of individual with whom he is dealing. Individuals with irregular sleeping and eating habits, and sedentary individuals may be harder to bring under allergic control than otherwise. Excessive smoking, coffee drinking and indulgence in alcoholic beverages may also interfere with allergic management.

Psychiatric History (Questions 317 through 367)

It is well known that feelings, emotions and moods are expressed in bodily change via the autonomic and somatic nervous systems and the glands of internal secretion. If normal, they can be of great service to the individual; if excessive or perverted, they become useless and generate harmful bodily response.

It should be emphasized that the answers to these questions may suggest the presence of a psychological disturbance, but it does not necessarily follow that this disturbance is the entire cause of the allergic symptoms. When both allergic symptoms and psychological disturbances occur in the same individual, the theoretical possibilities are as follows: (1) the allergy may cause the nervous symptoms, (2) the psychic disturbance may be the only cause of the allergy, and (3) one condition may aggravate the other. The latter is the most frequent occurrence.

TECHNIQUE OF USING THE SUMMARY SHEET OR CARD

When the Allergy History Questionnaire has been returned by the patient, the physician should transfer the essential information obtained therefrom to the Summary Sheet or Card by circling the indicated headings or numbers on the latter. This form is then incorporated into the patient's chart with a great conservation of space. In addition, the physician is thereby enabled to quickly review the history whenever the patient returns to the office.

After the history portion of the Summary Sheet or Card has been completed, it is recommended that a Provisional Program be planned. This is most important because it ensures a complete work-up for the patient and lessens the likelihood that certain desirable studies would be overlooked. In this connection, it should always be remembered that the results of treatment are directly proportional to the thoroughness of the diagnostic investigation.

As we mentioned previously, the physician himself should review every Allergy History Questionnaire and plan a Provisional Program; it is not fair to the patient to delegate this task to an office nurse or secretary, for these individuals, no matter how efficient, do not have sufficient medical background to know what areas of the history need more investigation, what confirmatory or diagnostic studies should be done or what consultations are necessary. In our hands, less than five minutes are required to study the Allergy History Questionnaire, check the questions which need further investigation, transfer the pertinent information to the Summary Sheet, and plan a Provisional Program. If the physician does not want to devote this minimum of time to his patient, it is unlikely that he should be practicing allergy.

REFERENCES

1. Anderson, J. R., and Rubin, W.: The allergic load, Arch. Otolaryng., 58:540-545 (Nov.) 1953.

ALLERGY HISTORY QUESTIONNAIRE SUMMARY SHEET

NAME _____ ADDRESS _____ AGE _____

OCCUPATION _____ Single, Married, Separated, Divorced, Widowed

HISTORY:

Season: Spring, Summer, Autumn, Winter

Symptoms: Nose, Throat, Ears, Eyes, Chest, G-I, Skin, Miscellaneous, Bacterial, Headaches, Tiredness

Childhood History: Positive Negative

Family History: Positive Negative

Non-Pollen Inhalant History:

House Dust	Mold
Feathers	Paper
Smoke	Orris Root
Insecticides	Cottonseed-Flax.
Animal Danders	

Food History: Positive Negative

Physical History: Positive Negative

Focal Infection: Positive Negative

General Medical Survey:

1. Eyes	11. Kidneys
2. Nose	12. Blood
3. Mouth	13. Diabetes
4. Throat	14. Injury
5. Ears	15. Nervousness
6. Thyroid	16. V. D.
7. Chest	17. Serious operation
8. Heart	18. Endocrine
9. G. B. & Liver	19. Prostate
10. G-I	

Social History:

Smoking	Alcohol
Coffee	Nutrition

Psychiatric History:

Inadequacy	Anger
Sensitivity	Anxiety
Depression	Tension

PROVISIONAL DIAGNOSIS:

1. Seasonal Hay Fever
2. Seasonal Rhinitis
3. P. N. A.
4. Bronchial Asthma

PROVISIONAL PROGRAM:

1. Nasal Smear
2. Inhalant tests (1/100 T, 1/10 T, 1/1 T)
3. Food tests
4. Autogenous vaccine
5. X-ray sinuses
6. X-ray chest
7. C. B. C.
8. Urinalysis
9. Kahn
10. B. M. R.
11. Blood cholesterol
12. Blood sugar
13. Glucose tolerance
14. Blood calcium
15. Blood pressure
16. Nasopharyngoscopy
17. Indirect laryngoscopy
18. Audiometry
19. Caloric test
20. Eye consultation
21. Dental consultation
22. Medical consultation
23. Neurological consultation
24. Psychiatric consultation
25. Gynecological consultation
26. Urological consultation
27. Diet Survey
28. Bronchogram
29. Bronchoscopy
30. Surgery
31. Sinus wash
32. Diet survey
33. Dust-free instructions
34. Eliminate smoking
35. Eliminate alcohol
36. Eliminate coffee
37. Eliminate pets
38. Other

1a. ———: The diagnosis and treatment of upper respiratory symptoms produced by increased viscosity of mucus and dryness of the mucous membranes, Laryngoscope, 66: 85-92 (Jan.) 1956.

2. Brodman, K.; Lodge, I.; Erdmann, A. F., Jr., and Wolff, H. G.: The Cornell Medical Index Health Questionnaire, New York, Cornell University Medical College.

3. Hansel, French K.: Clinical Allergy, St. Louis, Mosby Co., 1953.

4. Rinkel, H. J.; Randolph, T. G., and Zeller, M.: Food Allergy. Springfield, Ill., Charles C Thomas, 1951.

Date_____ History Number_____

ALLERGY HISTORY QUESTIONNAIRE

Print Your Name_____ Print Your Address_____ Print Your Age_____

Circle whether you are: single married separated divorced widowed

What is your occupation?_____ For how long?_____

Circle the Highest Year You Reached in School:

Elementary: 1 2 3 4 5 6 7 8
High School: 1 2 3 4
College: 1 2 3 4

What is your main complaint?_____

How long have you been suffering with this?_____

DIRECTIONS:

If you can answer YES to the question asked, put a circle around the (Yes)

If you have to answer NO to the question, put a circle around the (No)

Answer every question you can.

If the patient is a child, the parent should answer as best he can for the child.

SEASONAL INCIDENCE

Do you have trouble, or is your condition worse

1. In the spring? Yes No
2. In the summer? Yes No
3. In the fall? Yes No
4. In the winter? Yes No

HISTORY OF ONSET OF CONDITION

5. Did your trouble begin gradually? Yes No
6. Did your trouble begin suddenly? Yes No
7. Do you have any idea of what caused the trouble to begin? Yes No

DISCUSSION OF ATTACKS

8. Do the attacks begin gradually? Yes No
9. Do the attacks begin suddenly? Yes No
10. Do you have attacks more than
    Once a day? Yes No
    Once a week? Yes No
    Once a month? Yes No
    Once a year? Yes No
11. Do the attacks usually last
    just a few minutes? Yes No
    several hours? Yes No
    several days? Yes No
    a whole season? Yes No
    the whole year long? Yes No
12. Do the attacks occur regularly at a particular time of the day or night? Yes No
13. Do you have any idea what brings on the attacks? Yes No
14. Have you found anything which will relieve the attacks? Yes No
15. Are the attacks serious enough to interrupt your daily routine or to make you lose sleep? Yes No

GENERAL ALLERGIC SYMPTOMATOLOGY

A. NOSE SYMPTOMS

16. Does the inside of your nose often itch? Yes No
17. Do you have attacks of sneezing, particularly on arising in the morning? Yes No
18. Does your nose block often? Yes No
19. Do you have headaches when your nose blocks? Yes No
20. Do you have to blow mucus from your nose often? Yes No
21. Are you bothered with mucus dripping from the back of your nose into your throat? Yes No

22. If you are bothered with a post-nasal discharge, is it foul-tasting (tastes badly)? ..... Yes No
23. Have you ever had polyps removed from your nose? ..... Yes No
24. Have you ever been treated for "sinus trouble"? ..... Yes No
25. Do you have hay fever? ..... Yes No
26. Is your sense of smell normal? ..... Yes No
27. Do you seem to get frequent colds? Yes No
28. When you catch cold, does it seem to hang on for a long time? ..... Yes No

B. THE THROAT

29. Do you frequently have itching of the palate or back part of the throat? ..... Yes No
30. Do you often have sore throat in the morning which passes off during the day? ..... Yes No
31. Do people tell you that you snore or sleep with your mouth open? Yes No
32. Do you have to clear mucus from your throat in the morning upon awakening? ..... Yes No
33. Do you have to clear your throat frequently? ..... Yes No
34. Do you get hoarseness or huskiness of the voice at times even though you have no cold? ..... Yes No
35. Do you get ulcers in your mouth? .... Yes No
36. Does your tongue feel swollen, sore, or appear excessively reddened at times? ..... Yes No
37. Do your lips swell at times? ..... Yes No

C. THE EARS

38. Are you bothered with itching of the ear canals? ..... Yes No
39. Do you have frequent attacks of ear pain? ..... Yes No
40. Do you sometimes feel as though there is a drop of water in your ear? ..... Yes No
41. Are you bothered with attacks of hearing loss in either ear which later clears up? ..... Yes No
42. Do you have attacks of dizziness? Yes No
43. Do you have noises in the ears from time to time? ..... Yes No
44. Do you have chronic drainage from either of your ears? ..... Yes No

D. THE EYES

45. Are you bothered with itching of the eyes? ..... Yes No
46. Do your eyes water often? ..... Yes No
47. Do your eyes become irritated at times? ..... Yes No
48. Do you often suffer with "blood-shot" eyes? ..... Yes No
49. Does the skin of your eyelids become reddened and swollen? Yes No
50. Do the margin of your eyelids become reddened and swollen? Yes No
51. Do you get eczema about your eyes? ..... Yes No
52. Does the light seem to hurt your eyes? ..... Yes No
53. Do your eyes sometimes have a thick ropy secretion which causes the lids to stick together in the morning? ..... Yes No

E. THE CHEST

54. Do you cough frequently? ..... Yes No
55. Do you have asthma? ..... Yes No
56. Do you have frequent attacks of bronchitis? ..... Yes No

F. GASTROINTESTINAL SYSTEM

57. Do you frequently belch after meals? ..... Yes No
58. Do you often have indigestion following meals? ..... Yes No
59. Is there any food which you feel disagrees with you each time you eat it? ..... Yes No
60. Do you experience periods when your appetite is poor? ..... Yes No
61. Do you often have attacks of diarrhea? ..... Yes No
62. Do you ever pass mucus in your stools as far as you know? ..... Yes No
63. Do you suffer with constipation? ..... Yes No
64. Do you suffer with cramping pains in your lower abdomen? ..... Yes No
65. Have you ever been told you have chronic appendicitis? ..... Yes No
66. Have you ever been told you have mucous colitis or colic? ..... Yes No
67. Have you ever been told you have gallbladder disease? ..... Yes No
68. Have you ever had acute pain in the abdomen associated with hives and itching skin? ..... Yes No

G. THE SKIN

69. Do you suffer with eczema or any other chronic skin eruption? ..... Yes No
70. Do you sometimes suffer with hives? Yes No

71. Are you bothered with itching of the skin? Yes No
72. Do you sometimes get swelling of certain parts of the skin for no apparent reason? Yes No
73. Does contact of your skin with certain substances cause a rash or "breaking out"? Yes No
74. Have you ever had poison ivy eruption? Yes No
75. Have you ever had a rash from any drug you took by mouth, applied to your skin, or was given you by needle? Yes No
76. Does your skin blister easily? Yes No
77. Do you perspire a lot? Yes No

H. MISCELLANEOUS SYMPTOMS

78. Do you have frequent headaches? Yes No
79. Are there times when all you want to do is sleep, when thinking correctly and concentrating is difficult? Yes No
80. Do you have convulsions? Yes No
81. Do you seem to be tired more often than most people? Yes No
82. Do you suffer with "rheumatism" of the joints or muscles? Yes No
83. Did you ever get chills, fever, enlarged glands or joints, or a skin rash after the injection of serum for lockjaw, diphtheria, etc. Yes No
84. As far as you know, are you sensitive to any of the following drugs: aspirin, anacin, B-C, Stanback, sulfa drugs, penicillin, Aureomycin, Terramycin, quinine, idodine, nose drops, laxatives, sedatives, codeine, morphine, Demerol, or any other drug? Yes No

CHILDHOOD ALLERGIC HISTORY

(for adults only)

As far as you know, when you were a child:

85. Did you have eczema or any other chronic skin trouble? Yes No
86. Did you have colic? Yes No
87. Were you a feeding problem? Yes No
88. Were you often sick? Yes No
89. Did you gain weight normally? Yes No
90. Did you have frequent colds? Yes No
91. Did you have frequent attacks of sore throat? Yes No
92. Did you have sinus trouble? Yes No
93. Did you have frequent ear abscesses? Yes No
94. Did you have asthma? Yes No
95. Did you have bronchitis? Yes No
96. Did you have croup? Yes No
97. Did you have persistent coughs? Yes No
98. Did you have hay fever? Yes No
98-a Did you have frequent attacks of "stomach-ache," diarrhea, or vomiting? Yes No

FAMILY HISTORY

Have any members of your family had any of the following diseases? This includes your mother, father, grandparents, aunts, uncles, brothers, sisters, and children (if any).

99. Asthma? Yes No
100. Hay Fever? Yes No
101. Nasal allergy? (frequent attacks of sneezing, running nose, blockage of the nose, postnasal drip) Yes No
102. Hives? Yes No
103. Eczema? Yes No
104. Chronic skin disease? Yes No
105. Frequent headaches? Yes No
106. Migraine? Yes No
107. Chronic indigestion? Yes No
108. Colitis? Yes No
109. Low blood pressure? Yes No
110. Epilepsy? Yes No

NON-POLLEN INHALANT HISTORY

(HOUSE DUST)

Do you notice that your trouble begins or is aggravated

111. When the house is being cleaned or swept? Yes No
112. When rugs are being cleaned? Yes No
113. When the bed is being made or the mattress is being turned? Yes No
114. During spring house cleaning? Yes No
115. When the first cold snap of autumn comes? Yes No
116. In such dusty places as: theatres, churches, grocery stores, department stores, libraries, your bedroom? Yes No

(ATMOSPHERIC MOLD)

Do you notice that your trouble begins or is aggravated

117. During prolonged periods of damp weather? Yes No

118. When you are around where grass is being mowed or weeds are being cut? ..... Yes No

119. When you are near hay or straw (as at the circus, in a barn, around a hay stack, on a hay ride)? ..... Yes No

120. When you go into an old damp house, a damp basement, shed, or cellar? ..... Yes No

121. When you enter a closet in which are stored old shoes, unused luggage, gloves, or other leather goods? ..... Yes No

122. If you eat cheese, mushrooms, cantaloupe, or drink beer? ..... Yes No

123. When you sit on old, overstuffed furniture? ..... Yes No

(FEATHERS)

Do you notice that your trouble begins or is aggravated

124. When lying on a feather pillow? ..... Yes No

125. When fluffing pillows ..... Yes No

126. If you use a down comfort? ..... Yes No

127. If you are near chickens, ducks, geese, pigeons, parrots, turkeys, canaries, or other birds? ..... Yes No

128. If you are around anyone who works around poultry or other fowl? ..... Yes No

(PAPER)

Do you notice that your trouble begins or is aggravated

129. When you use Kleenex or a similar type tissue? ..... Yes No

130. When you first open a folded newspaper? ..... Yes No

131. When you are around a lot of cut paper? ..... Yes No

(SMOKE)

Do you notice that your trouble begins or is aggravated

132. When you smoke? ..... Yes No

133. When you are around those who are smoking? ..... Yes No

134. When in night clubs or other smoky places? ..... Yes No

(ORRIS ROOT)

Do you notice that your trouble begins or is aggravated

135. When using face, talcum, body, bath, or tooth powder? ..... Yes No

136. In beauty parlors or barber shops? Yes No

137. When you are around people who use a lot of powder or perfume? Yes No

(PYRETHRUM-DERRIS ROOT-LETHANE-PARADICHLOROBENZENE)

Do you notice that your trouble begins or is aggravated

138. When you are exposed to household insect powder or sprays? ..... Yes No

139. When you are exposed to powders, sprays, or crystals used for moth-proofing purposes? ..... Yes No

140. When you are exposed to dusting powders or sprays used in the garden or on crops? ..... Yes No

(COTTONSEED-FLAXSEED)

Do you notice that your trouble begins or is aggravated

141. When you handle, or are around, animal or poultry feeds? ..... Yes No

142. When you use certain hair wave-sets, shampoos, or tonics? ..... Yes No

(ANIMAL HAIRS AND DANDERS)

Do you notice that your trouble begins or is aggravated

143. When you are around any of the following animals: dogs, cats, horses, goats, rabbits, cows, hogs, or sheep? ..... Yes No

144. When you handle or come into contact with any of the following: furs, rugs, certain articles of clothing, dress goods, blankets, gloves, hats, toy animals, or brushes? ..... Yes No

FOOD HISTORY

145. Do you suspect any food of causing or aggravating your condition? Yes No

146. Are there any foods which you dislike? ..... Yes No

147. Are there any foods which you overindulge in or eat frequently because you like them so much? Yes No

148. Is there any seasonal food (for example, strawberries) which you overindulge in? ..... Yes No

149. Are there any foods which you find difficult to digest? ..... Yes No

150. Do any foods you eat cause nausea, vomiting, diarrhea, heartburn, belching, gas on the stomach, cramps, hives, skin rashes, headache? ..... Yes No

151. Are you on any type of diet at present? ..... Yes No

PHYSICAL HISTORY

Does your trouble at times seem to begin or become aggravated

152. By changes in the weather? Yes No
153. By damp, humid weather? Yes No
154. By being exposed to draughts? Yes No
155. When you get your feet wet? Yes No
156. When you become chilled? Yes No
157. By exercise? Yes No
158. By fatigue? Yes No
159. By loss of sleep? Yes No
160. By excitement? Yes No
161. By emotional upheaval? Yes No
162. By hot or cold baths? Yes No
163. By becoming overheated? Yes No
164. By prolonged periods of physical or mental work, prolonged strain (as when there is someone sick in the family)? Yes No
165. As a result of "nervousness"? Yes No
166. Air conditioning? Yes No

FOCAL INFECTION HISTORY

167. Are you conscious of a foul odor in your nose? Yes No
168. Do you have a dripping from the back of your nose into your throat which has a "sickening sweet" taste or is yellow or green like pus? Yes No
169. Do you have any bad teeth in your mouth? Yes No
170. Do your gums bleed? Yes No
171. Do you have bad tonsils? Yes No
172. Do your ears drain? Yes No
173. Have you been told that you have gall bladder trouble? Yes No
174. Do you have increased frequency of urination? Yes No
175. Does urinating cause a burning sensation? Yes No
176. As far as you know, do you have pus in your urine? Yes No
177. Are you bothered with a genital dicharge? Yes No

(For Women Only)

178. Have you been told you have a laceration or erosion of your womb or that you need to be cauterized? Yes No

(For Men Only)

179. Have you ever been told that you have infection in your prostate gland? Yes No
180. As far as you know, do you have infection in any part of your body that we have not inquired about? Yes No

ENVIRONMENTAL SURVEY

(HOME SURVEY)

181. Is your house old? Yes No
182. Is your house new? Yes No
183. Is your house damp? Yes No
184. Is your house dry? Yes No
185. Do things mildew easily around the house? Yes No
186. Near your house, is there
a factory? Yes No
a railroad? Yes No
a lake? Yes No
a barn? Yes No
a poultry yard? Yes No
a swampy area? Yes No
lots of weeds? Yes No
lots of trees? Yes No
anything you suspect as being a possible cause of your symptoms? Yes No
187. Is your house heated by
open gas heaters? Yes No
radiators? Yes No
floor gas furnaces? Yes No
open fire places? Yes No
central heating system with ducts? Yes No
188. Is your house cooled by an attic fan? Yes No
window air-conditioning? Yes No
central air-conditioning? Yes No
window fans? Yes No
189. Do you have plants in the house or in window planter boxes? Yes No
190. Do you have pets in the house or in the yard? Yes No
191. Is the house cleaned with
broom and mop? Yes No
vacuum sweeper? Yes No
192. Do you use insect sprays or powders or moth repellants in the house? Yes No
193. Do you keep any books or magazines that gather dust in the house? Yes No
194. Do you have pictures on the wall? Yes No
195. Do you have overstuffed furniture? Yes No
196. Do you use cotton mattresses? Yes No
197. Do you use feather pillows? Yes No
198. Do you use down comforts on the bed? Yes No
199. Do you have rugs on the floor? Yes No
200. Do you use padding under your rugs? Yes No

201. Do you have draperies on the windows? Yes No
202. Do you have any stuffed birds or like trophies in the house? Yes No
203. Do you have throw pillows around the house? Yes No
204. Are there any fumes or smells continuously or frequently present about the house? Yes No
205. Is there any place in the house where you have symptoms regularly? Yes No
206. Are the walls of your house covered with wall paper? Yes No
207. Is any type business enterprise carried out in your home? Yes No
208. Are you engaged in any hobby in your home? Yes No

(WORK OR SCHOOL SURVEY)

209. Do you have symptoms at work or school? Yes No
210. Is the place where you work
damp? Yes No
cooled wth window or attic fans? Yes No
air-conditioned? Yes No
centrally heated? Yes No
dusty? Yes No
smoky? Yes No
211. As far as you know, do you inhale anything at work which might cause or aggravate your symptoms? Yes No
212. Are there fumes, gases, smokes, or odors where you work? Yes No
213. Do you come into contact with animals or use animal hairs at work? Yes No
214. Do you come into contact with grain dusts, animal feeds, paints, varnishes, sawdust, or powders at work? Yes No
215. Are insecticides used at work? Yes No
216. Do you frequently perspire profusely at work? Yes No

(ACTIVITIES)

217. Do you take part in any outdoor sports? Yes No
218. Do you have any outdoor hobbies? Yes No
219. Are you better when you are away from home on a trip, such as at vacation time? Yes No

PREVIOUS TREATMENT HISTORY

220. Have you ever received treatment for your condition before? Yes No
221. Have you ever had any operations for your condition? Yes No
222. Have any of the treatments or drugs prescribed given prolonged relief? Yes No
223. Have you been treated or tested for allergy before? Yes No
224. At the present time, are you taking medicine for your condition? Yes No

GENERAL MEDICAL HISTORY

225. Do you often have pain in the face? Yes No
226. Do you often experience flushing of the face with a sensation of warmth? Yes No
227. Do you need glasses to read or see things at a distance? Yes No
228. Do you often suffer with pains in the eyes? Yes No
229. Have you ever had an operation on your eyes? Yes No
230. Did a doctor ever say you have trouble with your eyes? Yes No
231. Do you experience a sense of burning or dryness in the nose; that is a sense of stuffiness rather than actual blockage? Yes No
232. Do you often have pain inside your nose? Yes No
233. Are you bothered wth a bad odor in your nose? Yes No
234. Do you frequently blow large dried crusts or scabs from your nose? Yes No
235. Have you ever had severe nose bleeds? Yes No
236. Have you ever fallen or been hit hard on your nose? Yes No
237. As far as you know, was your nose ever broken? Yes No
238. Have you ever been told that you have a crooked bone in your nose? Yes No
239. Have you ever had an operation on nose or sinuses? Yes No
240. Are you bothered wth soreness or burning in your mouth? Yes No
241. Do you have bad breath at times? Yes No
242. Are you often bothered with a sensation of dryness in your mouth? Yes No
243. Do you have false teeth? Yes No
244. Is your tongue often sore? Yes No
245. Is your tongue often coated? Yes No
246. Do you often find yourself grinding your teeth, or do people tell you that you grind your teeth when you are sleeping? Yes No
247. Are you able to open your mouth wide? Yes No
248. Do you hear a clicking sound when you open your mouth wide? Yes No
249. Do you often have pain around your ears when you chew? Yes No
250. Do the glands in your neck swell when you get sore throat? Yes No
251. Have your tonsils been removed? Yes No

252. Do you often feel a lump, pressure, or tightness in your throat? Yes No
253. Do you have difficulty hearing? Yes No
254. Is your hearing worse while you are eating? Yes No
255. Do people frequently say you speak too loud? Yes No
256. Do people frequently ask you to repeat? Yes No
257. When people speak to you, can you hear them but have trouble making out what they are saying? Yes No
258. Have you ever had an abscess in your ears? Yes No
259. Have you ever had a fungus infection or sand boil in your ears? Yes No
260. Do you get a sense of fullness in your ears? Yes No
261. Do you have dandruff? Yes No
262. Do you have any swelling in the neck at present? Yes No
263. Did a doctor ever tell you that you have a goiter? Yes No
264. Did a doctor ever give you thyroid extract to take? Yes No
265. Dd you ever have a basal metabolism (breathing) test? Yes No
266. Have you ever coughed up a large amount of blood? Yes No
267. Do you have any chronic chest trouble (except asthma)? Yes No
268. Did you ever have T.B. (tuberculosis)? Yes No
269. Do you live with anyone who has ever had or now has T.B.? Yes No
270. Did you ever have a chest or lung operation? Yes No
271. Did a doctor ever say you have heart trouble? Yes No
272. Do you have high blood pressure? Yes No
273. Do you have low blood pressure as far as you know? Yes No
274. Do your hands or feet ever swell badly? Yes No
275. Do you get short of breath easily? Yes No
276. Does your heart ever beat very fast or so hard it seems to shake your chest? Yes No
277. Do your hands or feet often feel cold even in hot weather? Yes No
278. Do you frequently have cramps in your legs? Yes No
279. Is your appetite poor? Yes No
280. Have you lost weight lately? Yes No
281. Do you gain weight easily? Yes No
282. Have you ever had ulcers in the stomach? Yes No
283. Did a doctor ever say you had gallbladder or liver trouble? Yes No
284. Did you ever pass blood in your bowel movements? Yes No
285. Did a doctor ever say you had kidney trouble? Yes No
286. Do you have rheumatism? Yes No
287. Did you ever have rheumatic fever? Yes No
288. Are you troubled with your feet? Yes No
289. Do you seem to get tired faster than the average person? Yes No
290. Are you sick often? Yes No
291. Did a doctor ever say you were anemic? Yes No
292. Do you have sugar diabetes? Yes No
293. Have you ever been seriously injured? Yes No
294. Do you consider yourself a nervous person? Yes No
295. Have you ever been treated for your nerves? Yes No
296. Have you ever had a nervous breakdown? Yes No
297. Did you ever have a fit or convulsion? Yes No
298. Have you ever been treated for bad blood (venereal disease)? Yes No
299. Have you ever had a serious operation? Yes No
300. Are you under any other doctor's care at the present time? Yes No

(For Women Only)

301. Are your menstrual periods usually painful? Yes No
302. Are your periods regular? Yes No
303. Have you had any female operations? Yes No
304. Are you changing life at the present time? Yes No
305. Have you finished changing life? Yes No
306. Are you taking hormone shots at the present time? Yes No
307. Are you pregnant at the present time? Yes No

(For Men Only)

308. Do you have trouble starting your stream when urinating? Yes No

SOCIAL HISTORY

309. Do you usually have great difficulty in falling asleep or staying asleep? Yes No
310. Do you find it impossible to take a regular rest period each day? Yes No
311. Do you find it impossible to take regular exercise? Yes No
312. Do you smoke more than 20 cigarettes a day? Yes No
313. Do you drink more than six cups of coffee or tea a day? Yes No

314. Do you usually take two or more alcoholic drinks a day? Yes No
315. Do you usually eat in between meals? Yes No
316. Do you often skip meals entirely? Yes No

INADEQUACY

317. Do you sweat or tremble a lot during examinations or questioning? Yes No
318. Do you get nervous and shaky when approached by a superior? Yes No
319. Does your work fall to pieces when the boss or a superior is watching you? Yes No
320. Does your thinking get completely mixed up when you have to do things quickly? Yes No
321. Must you do things very slowly in order to do them without mistakes? Yes No
322. Do you always get directions and orders wrong? Yes No
323. Do strange places or people make you afraid? Yes No
324. Are you scared to be alone when there are no friends near you? Yes No
325. Is it always hard for you to make up your mind? Yes No
326. Do you wish you always had some one at your side to advise you? Yes No
327. Are you considered a clumsy person? Yes No
328. Does it bother you to eat anywhere except in your own home? Yes No

DEPRESSION

329. Do you feel alone and sad at a party? Yes No
330. Do you usually feel unhappy and depressed? Yes No
331. Do you often cry? Yes No
332. Are you always miserable and blue? Yes No
333. Does life look entirely hopeless? Yes No
334. Do you often wish you were dead and away from it all? Yes No

ANXIETY

335. Does worrying continually get you down? Yes No
336. Does worrying run in your family? Yes No
337. Does every little thing get on your nerves and wear you out? Yes No
338. Are you considered a nervous person? Yes No
339. Does nervousness run in your family? Yes No
340. Did you ever have a nervous breakdown? Yes No
341. Did anyone in your family ever have a nervous breakdown? Yes No
342. Were you ever a patient in a mental hospital (for your nerves)? Yes No
343. Was anyone in your family ever a patient in a mental hospital (for their nerves)? Yes No

SENSITIVITY

344. Are you extremely shy or sensitive? Yes No
345. Do you come from a shy or sensitive family? Yes No
346. Are your feelings easily hurt? Yes No
347. Does criticism always upset you? Yes No
348. Are you considered a touchy person? Yes No
349. Do people usually misunderstand you? Yes No

ANGER

350. Do you have to be on your guard even with friends? Yes No
351. Do you always do things on sudden impulses? Yes No
353. Do you go to pieces if you don't constantly control yourself? Yes No
354. Do little annoyances get on your nerves and make you angry? Yes No
355. Does it make you angry to have anyone tell you what to do? Yes No
356. Do people often annoy and irritate you? Yes No
357. Do you flare up in anger if you can't have what you want right away? Yes No
358. Do you often get into a violent rage? Yes No

TENSION

359. Do you often shake or tremble? Yes No
360. Are you constantly keyed up and jittery? Yes No
361. Do sudden noises make you jump or shake badly? Yes No
362. Do you tremble or feel weak whenever someone shouts at you? Yes No
363. Do you become scared at sudden movements or noises at night? Yes No
364. Are you often awakened out of your sleep by frightening dreams? Yes No
365. Do frightening thoughts keep coming back in your mind? Yes No
366. Do you often become suddenly scared for no reason? Yes No
367. Do you often break out in cold sweats? Yes No

VASCULAR AND ALLERGIC HEADACHES

A Panel Discussion

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RAYMOND L. HILSINGER, M.D.
CINCINNATI, OHIO
BERNARD M. BARRETT, M.D.
PENSACOLA, FLORIDA

DR. HANSEL: Although the problem of vascular headaches was discussed at a previous meeting of this Society in 1952, I think that it is worth while to review some of the advances that have occurred in the interim.

We are concerned mostly with the common types of vascular headaches which are encountered in otolaryngologic practice. In the literature in general most reference to headache is under the term of migraine. There is a tendency to include all the different types of headache in a few groups of the migraine type. It has been our experience that the incidence of migraine is not more than 2 to 3 per cent. The type of headache which we encounter in otolaryngology may be somewhat different from that noted by the neuropsychiatrist, the allergist, or the internist. Patients consult us chiefly when the headache is localized in the region of the sinuses. It is common for a patient to complain that he has "sinus," and I say, "What do you mean by 'sinus'? Do you have a stuffy nose?"

"Yes."

"Does your nose run?"

"Yes."

The first impression is that the patient is allergic, but as the history is compiled the pattern typical of the vascular type of headache often evolves.

About 1941 Horton's paper entitled "The Use of Histamine in the Treatment of Specific Types of Headaches" appeared in the Journal of the American Medical Association. About that time we had a patient who complained of pain over the antrum; the dentist had removed a tooth, so it seemed to us that we should investigate the possibility of an antrum infection. Smear tests and roentgenograms did not reveal any infective organism or pathologic changes; a detailed history was not taken. Before the patient's next visit, we had read Horton's paper and had become familiar with the new syndrome. When the patient reported for the next visit, we obtained a more detailed history, and it revealed a perfectly classical case of vascular headache involving the forehead, the eye, the cheek, and the upper teeth. Many of these patients have pain in the teeth and in the antrum region. Stuffiness of the nose on the involved side is very common. Sometimes a patient has used nose drops to relieve that stuffiness. Sometimes the stuffiness will precede the onset of an attack, or it may be present without the headache. The relief of this stuffiness by benzedrine or some local vasoconstrictor sometimes will prevent the attack from coming on.

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The pattern Horton described, a so-called vasodilator type, is one of quick onset, occurring often during the night. The patient awakens from sound sleep with a terrible pain in the region of the eye, the forehead and the teeth. He may arise from bed and walk around, and the activity may restore vascular balance and relieve the attack. Symptomatic drugs may be employed also for immediate relief.

Now, this represents a rather typical pattern in that it is cyclic in nature. The patient may report that he had had these headaches for two or three years, then for two or three years he had no headaches; or that they occurred at certain times of the year, such as in March or in October. It is characteristic of this type of headache that during the headache cycle, attacks may be precipitated by the smallest amount of alcohol; during the headache-free cycle, alcohol may be ingested without precipitating an attack. Many variations of the pattern may occur. In general, these patients are not of the so-called tense type. Some of these patients, especially the typical ones, are really placid, quiet individuals. They sleep very soundly at night. In women the tendency is for the headache to come on late in the night, almost in the early morning. In men it comes on earlier in the night.

(Slide) Bear in mind that the pain of vascular headache may occur in any of the arteries in the head and neck, and is not restricted to those in the region of the antrum, the eye, and the forehead. In some patients the pain first appears in the frontal and ocular areas. In others the occipital area or any of the branches of the temporal arteries or deep cervical branches may be primarily involved. We noted a very interesting group of cases in which the involvement appeared in the thyroid or laryngeal arteries. In these instances the patients had rather typical globus hystericus symptoms. It is important to note that pain similar to that involving vessels in the head may also affect other vessels, perhaps in the arm or in the leg. The pattern may shift from one vessel to another or from one side to the other.

We have found that the symptom pattern of globus hystericus so frequently seen when one of the laryngeal arteries, the thyroid artery, or the cricothyroid artery is involved, may correspond to the type of headache with quick onset, to the tension type of headache, or to the more prolonged attacks of pain which may occur whether the patient is tense or not tense. The symptoms correspond in their course with those of the true headaches.

Finally, we shall summarize treatment as related to the use of histamine, and the administration of various drugs for relief.

Dr. Hilsinger, I should like for you to point out in your discussion what doses of histamine you use in treatment.

DR. HILSINGER: The headache patient has an unstable autonomic nervous system, the center of which is located in the hypothalamus. The patient's autonomic nervous system, being a composite of those of his ancestors, is geared to a certain rate of speed. When this rate is exceeded under the stress of modern living, imbalance between the sympathetic and parasympathetic divisions of this system occurs. This results in autonomic dysfunction, which expresses itself in this patient as a vasodilating or vasospastic headache. The basic pathologic change is located somewhere in the carotid arterial tree, either in the vasa vasorum of the walls of the larger blood vessels or in the end arteries of the small blood vessels. The microscopic pathologic change is at the functional vascular level: the arteriolar-capillary-

venular unit. The shock organ of the headache patient is located then, in the walls of the blood vessels. This shock organ is located in the inner ear in Ménière's disease; in the bronchial tube in asthma; and in the nasal mucosa in hay fever.

This condition is probably all a matter of degree. I like to compare it to a heart condition. If the condition is angina, it is probably akin to what we say is histaminic cephalalgia. If it is a thrombosis, even though partial, it is probably like migraine. For the ease of treatment, I would like to have a gross classification of headaches. I agree with Dr. Hansel that migraine is a very rare disease and that we see it in only about 2 or 3 per cent of cases.

Hilger believes, and I agree with him, that the arteriolar-capillary-venular unit is the functional vascular basis of all our problems (fig. 1). When something happens to cause stress, arteriolar spasm causes a cessation of blood flow, with resultant backflow from the venous side and extravasation of fluid from the capillary bed into the surrounding tissue (fig. 2).

For convenience in my personal practice, I class as vasodilating headaches, migraine, histaminic cephalalgia, tension headache and carotid headache. Hansel calls the carotid headache atypical histaminic cephalalgia. This headache results from pain in an end artery whereas the typical histaminic cephalagia is in the walls of blood vessels. Vasospastic headaches we call myalgia.

Both symptomatic and prophylactic therapy play a part in the treatment of vascular headaches. In the symptomatic treatment of vascular headache, we use cafergot and cafergot PB orally, the latter a comparatively new medication. For the patient who is unable to take oral medication, we use cafergot or cafergot

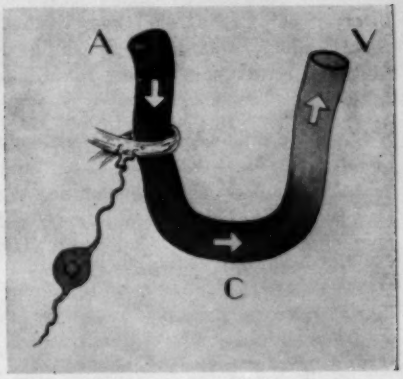

FIG. 1—The functional vascular level: the arteriolar-capillary-venular unit.

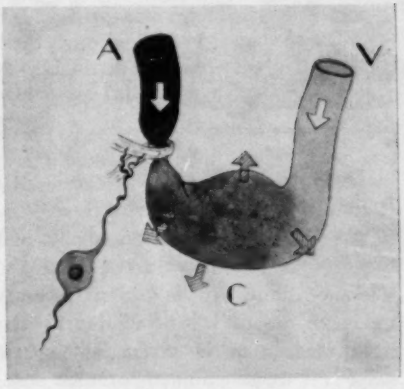

FIG. 2—Arteriolar spasm and its sequelae.

PB suppositories (the latter is used in cases where there is an associated tension); or parenteral medication with gynergen intramuscularly, or dihydroergotamine (DHE-45) intramuscularly or intravenously.

Other medications for tension headache are APC combined with the barbiturates and fiorinal. In the tension headaches, ergotamine is not indicated. In hangover headaches which have lasted three or four hours, ergotamines are no longer helpful, and I find that neocalglucon, 20 per cent, from 5 to 10 cc.

intravenously, is a marvelous medication. In myalgia due to vasospasm, nicotinic acid intravenously gives immediate relief.

In the specific prophylactic treatment of migraine, use small doses of nicotinic acid, or small doses of histamine. We start with dilution 5 or dilution 6 of histamine diphosphate (table I). The initial dose is .05 cc. intradermally, and the dose is gradually increased to the tolerance limit. If the patient receives too much, an attack is brought on; if too little medication is given, he is not helped; if we give him an optimum dose, he is immediately relieved. We also use some psychotherapy associated with rest and relaxation. The patient is advised to avoid emotional upsets, to eliminate faulty habits, and even to change his job, if this seems necessary.

In histaminic cephalalgia, we use histamine diphosphate in the same dosage.

I treat tension headache by helping in personality adjustment and using bellergal, one tablet fifteen minutes before meals and one tablet at bedtime.

In carotid headache, the treatment is the same as in migraine or histaminic cephalalgia.

The General Prophylactic Treatment of Vascular Headaches (Table II)

These people do not eat enough of the right kind of food, so an adequate nutritional regimen must be carried out. Massage is used, and we get some help from the masseur or from the osteopath. Loss of sodium is a big help to these people. A fever bath at night followed by a cold shower in the morning helps to stimulate the autonomic nervous system. The technique of a fever bath is as follows: The patient is given one or two capsules of copavin and is told to take a hot bath lasting twenty minutes. The patient spends the next hour in bed covered with several blankets, during which time he perspires profusely. He then takes a cool bath, changes to fresh nightclothes and retires for the night. Vitamin B and electrolytes, especially potassium and calcium, are given. Potassium is important because it helps make adrenalin work, and calcium because it helps the integrity of the capillary bed. Many of these patients, especially those with myalgia, have a low basal metabolic rate, and when this is true, thyroid therapy is helpful.

Steroids are of great help, especially meticorten and meticortelone. They are

TABLE I

TREATMENT OF HISTAMINIC CELPHALALGIA WITH HISTAMINE DIPHOSPHATE

Dilution No. 3	1:1000; 2.75 mg./1 cc.
Dilution No. 4	1:10,000; .275 mg./1 cc.
Dilution No. 5	1:100,000; .0275 mg./1 cc.
Dilution No. 6	1:1,000,000; .00275 mg./1 cc.
Dilution No. 7	1:10,000,000; .000275 mg./1 cc.
Dilution No. 8	1:100,000,000; .0000275 mg./1 cc.
Weaker dilutions if indicated	

TABLE II

GENERAL PROPHYLACTIC TREATMENT OF VASCULAR HEADACHE

Adequate nutritional regimen
Massage
Fever bath at night; cold shower in morning
Solu-B or folbesyn intravenously
Electrolytes: potassium, calcium. Thyroid, steroids, mineral, vitamin C
Nicotinic acid, intravenously and orally
Restriction-of-salt diet, if necessary

especially useful in histaminic cephalalgia if histamine desensitization does not bring relief.

My nine commandments for the headache patient follow:

1. This is not a perfect world. Families and friends have many foibles. Perfection is rarely attained, so be satisfied with less.

2. Tolerance makes understanding the other fellow easier. It sets an attainable standard.

3. Do not be a slave to the clock. Work at your own pace; do as much as you can. Trying to meet too many deadlines only creates tension.

4. You cannot please everybody, so stop trying. Popularity comes by giving your friends and family a chance to love you for yourself, not for your best performance.

5. Be efficient, yes, but not to the extent that perfection becomes a burden.

6. Speak up if you want to. You cannot please everybody; honesty and directness break down barriers and make friendships easier.

7. Approve of yourself. You are as good as the next fellow. If you are given a compliment, take it and make use of it.

8. Stop being so critical of your negative feelings. Everyone is ambivalent at times, so do not worry so much about loving and hating.

9. Last and most important: Stop feeling guilty. We are all human beings and we all make errors. Give a little and you will get a lot, maybe even a reduction of that pain in your head.

DR. BARRETT: In a recent paper before the New York Academy of Science, Dr. Hansel and I reported some preliminary observations on the use of reserpine as an adjunct in resistant headache patterns. At that time the number of cases was exceedingly small, but we are now in a position to report on an additional number.

First of all, by resistant headache patterns we mean headaches which persist regardless of etiologic evaluation, and proper therapy which has been so excellently demonstrated to you by Dr. Hilsinger. We find a few cases that just won't respond to conventional methods. We have classified them on the basis of vascularity, and essentially they fall in the categories Dr. Hilsinger has set up, except for the carotid type of headache, which we call atypical histaminic cephalalgia.

We have three additional cases of migraine to report on. This type of headache is extremely rare in my practice. By utilizing reserpine as an adjunct to other therapy, I have one patient who is having good results, one with only fair, and one which has not responded to this therapy.

In the category "histaminic cephalalgia" we have 14 additional cases to report. Six of these patients have responded well to resperine; six have shown fair results; and the results in two cases have been poor.

In the category which Dr. Hilsinger classifies as the carotid type, but which we call atypical, we have 18 additional cases treated with reserpine: 9 with good results; 6 with fair results; 3 with poor results. This atypical group includes patients who have had tenonitis, but consists predominantly of patients with globus hystericus.

Of 13 cases of headache due to vascular tension which received reserpine

therapy, 7 cases showed good results from the treatment, 4 had fair results, and in 2 cases the results were poor.

I don't want to confuse vascular-tension and muscular-tension headache. Muscle tension may precede a headache; it is frequently associated with the headache pattern; and it may persist for quite a period of time after the actual headache is gone.

Resperine is not a new drug. It has been used for over three hundred years in Europe in treatment of anxiety states. Its use in treating hypertension has been of comparatively recent origin. The pure product was isolated as recently as 1952.

In using reserpine, the dosage prescribed should be considered with respect to the nature of the case. The dosage prescribed for patients either with marked hypertension or with marked psychosis should be much larger than that employed for a patient with a vascular headache whose tension is essentially normal and who has no psychosis. We have found that the optimum dose is about a tenth of a milligram three times a day for a week. The dosage should then be increased or decreased, depending upon the patient's response. Eventually, a maintenance dose of a tenth of a milligram gives the most satisfactory results. The nature of the drug is such that it is very slow in making its therapeutic manifestations.

Many people experience nasal stuffiness or numbness of the extremities while taking even this moderate dosage of reserpine. They may also have some occasional feeling of weakness until the body gets accustomed to the drug. Dr. Hansel and I have therefore recommended, and it has worked out very well, that initially these patients be given small quantities of one of the amphetamine products to tide them over the period during which the body is getting accustomed to the drug.

I would like to emphasize that the cases reported here were cases of resistant headache which had not responded to any other form of treatment. After a prolonged period of therapeutic failure, there is a big element of emotional tension, which may perpetuate a normal headache pattern which initially may have responded to routine therapy. Reserpine merits your consideration in the treatment of these resistant cases.

ACTH AND CORTICOSTEROIDS

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THE main function of the pituitary adrenal axis is the maintenance of healthy tissue and cells (homeostasis). There is a normal, continuous secretion of ACTH and corticosteroids. Any stress increases these secretions. There are three known mechanisms whereby the pituitary hormone, ACTH, is increased: (1) the direct action of adrenalin, (2) a low blood level of corticosteroids, and (3) a state of stress in the tissues.

ACTH, in turn, stimulates the adrenal cortex to secrete corticosteroids. The chief corticosteroids secreted by the cortex are hydrocortisone and corticosterone. About 50 to 100 milligrams of hydrocortisone are produced by a normal adult in 24 hours. This glucocorticoid, hydrocortisone, is of utmost importance in maintaining homeostasis. However, in so doing, it may also produce many physiologic, and at times pathologic, reactions.

Time will allow me to discuss only a few of these actions. (The terms corticosteroids and corticoids will be used interchangeably to designate the glucocorticoids, such as hydrocortisone and cortisone.)

ANTI-INFLAMMATORY ACTION OF CORTICOSTEROIDS

Dr. Thomas Dougherty has stated, at the October 1955 meeting of the American Rhinologic Society, that the inflammatory reaction always takes place in the connective tissue. This fact is not generally recognized, but it is of great importance. Whether the inflammation is of allergic, infectious or traumatic origin, it is always in the connective issue.

Inflammation can be produced by any stress and is a chain reaction, set up and continued by substances released from cell damage. The cells primarily damaged are the fibroblasts. The severity of inflammation depends upon the inflammatory stimulus and the amount of corticoids available. It is a definite and well-accepted fact that the corticosteroids have marked anti-inflammatory action and that growth hormone and thyrotropic hormone enhance inflammation.

Dougherty[1] gives the following hypotheses as to possible mechanisms in corticoid anti-inflammatory action:

1. Decrease in the level of circulating fibrinogen, thus producing a decrease in the amount of fibrin at the site of inflammation.
2. Inhibition of hyaluronidase, thus decreasing the action of the spreading factor.
3. Inhibition of the formation of ground substance.
4. Shedding of mast cell granules with release of a heparin-like substance, which has antihyaluronidase action and which also binds histamine.

To this list could be added the action in maintaining a good capillary bed. Dougherty believes that all of these actions take place but that there may be a more fundamental anti-inflammatory phenomenon involving the action of the hormones on the many substances liberated from cell damage.

INFECTION AND CORTICOSTEROIDS

There is no question but that the glucocorticoids in *large* doses impair

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the host's resistance to many infections. There are numerous explanations for this. Thomas[4] states that there are at least six possibilities:

1. A direct effect on the infecting organism. There is no direct evidence that this takes place.
2. An effect on the neutrophils. Although the corticosteroids produce a systemic leukocytosis, fewer pus cells migrate to the area of local inflammation. There is slight evidence, but no definite proof, that the bactericidal properties of leukocytes are diminished. Some virus infections are also enhanced by *large* doses of corticoids, and since available evidence shows that neutrophils are not important in virus infections, the leukocyte hypothesis becomes somewhat untenable.
3. Influence in antibody formation. There is no definite evidence that this is a factor in humans.
4. Interference with the inflammatory reaction. The corticoid depression of tissue inflammation is an accepted fact, but, as noted above, the explanation is uncertain. At any rate, the anti-inflammatory theory does not explain the enhancement of virus infections, nor the enhancement of bacteria which are injected directly into the blood stream. In these two instances, there is no local inflammatory reaction. Of course, some cellular component present in the blood stream could be involved and the hormone could work on it here.
5. The effect on serological mechanisms, such as the nonspecific hyaluronidase inhibitor. This is a definite possibility.
6. Damage to the reticuloendothelial system. The macrophages in this system are for the purpose of clearing the blood stream of bacteria and particulate matter. There is some evidence that like certain colloids, cortisone blocks the cells of the reticuloendothelial system so that the macrophages then cannot clear the blood stream of infectious organisms.

EDEMA AND CAPILLARY PERMEABILITY

In the past it has been generally thought that most substances pass to and from the capillaries by way of the interendothelial cement substance. Renkin[3] and Pappenheimer[2] have shown that while this may be true of water, it is not true of oxygen, carbon dioxide and lipid-soluble substances. These penetrate mostly through the endothelial cells themselves. Since hydrocortisone aids in maintaining healthy endothelial cells, it is of physiologic importance in supplying oxygen and nutrients to cells of all structures.

Zweifach[5] states that the capillary wall consists of more than just the endothelial cells and the endothelial cement. It is a complicated structure consisting of (1) proteinaceous material covering the inner surface of the endothelial cells and cement, (2) endothelial cells, (3) interendothelial cement, (4) the very important pericapillary sheath, and (5) the surrounding connective tissue. The pericapillary sheath, which consists of dense ground substance and fibrils, is probably one of the most important of all structures. When it is firm, it prevents leakage from the capillaries. One of the actions of the corticoids is to keep the pericapillary sheath and ground substance firm; hence, the importance of these hormones in preventing edema.

Also, some of the swelling and edema in ground substance is produced by the action of the hyaluronidase on the ground substance. Hyaluronidase depolymerizes the ground substance and makes it swell and become more liquid. This takes place under most stresses, including infection and allergy. Since corticosteroids are antihyaluronidase factors, they help prevent edema by this mechanism.

At the meeting previously mentioned, Dougherty also stated that the minute capillaries do not supply anything directly to any cell in the body. All substances have to penetrate the ground substance of the connective tissue before reaching the cells of all of our important organs. Vice versa, metabolites and other substances must reach the circulation via the ground substance. Since one of the functions of the corticoids is the

maintenance of healthy ground substance, these hormones take on added significance.

GENERAL ASPECTS AS RELATED TO OTOLARYNGOLOGY

The contraindications for the use of corticosteroids are so well known that they will not be commented upon except to say that they apply in otolaryngology just as in any other field.

A low-salt diet is advisable. High-protein diet with the addition of potassium is usually not necessary unless rather prolonged treatment with reasonably high dosage is used. During corticosteroid therapy, an occasional injection of ACTH is indicated in order to stimulate the adrenal cortex. Gradual corticoid withdrawal is usually not necessary except when the dosage has been fairly high and prolonged. Intravenous ACTH gives the maximum stimulation of the adrenal cortex. When infection is involved, the appropriate antibiotic should be used. Wound healing is not stopped but merely inhibited by the corticosteroids. For local use, hydrocortisone is the hormone of choice.

The use of these hormones in large doses over long periods should not be recommended for most otolaryngologic conditions. One of the most serious problems in corticosteroid prolonged high-dosage therapy is the depression of the adrenal cortex. If we eliminate this kind of therapy, we greatly reduce this danger. However, in surgical cases, if there is any possibility of cortical depression from previous therapy, prophylactic corticoids should be given before and for a few days following surgical stress. Intravenous hydrocortisone and intravenous ACTH should be available for emergencies.

The author has had only slight expeperience with the newer glucocorticoids, meticorten and meticortelone. These synthetic hormones are very powerful, but they have only very slight mineral activity. If time shows that other side reactions are of minimal degree, they should be very useful. They should be of particular value when used in conditions in which the main problem is one of edema.

OTOLARYNGOLOGIC CONDITIONS IN WHICH THE CORTICOSTEROIDS ARE USEFUL

1. *Hay Fever*
   Particularly in severe cases of hay fever not controlled by ordinary methods and in localities where the season is short, the corticosteroids may be useful. Besides their use in systemic therapy, the local use of hydrocortisone in eye drops is effective. The local use of hydrocortisone intranasally is only mildly efficacious.

2. *Status Asthmaticus*
   In this serious condition, intravenous hydrocortisone or intravenous corticotropin may be a life-saving procedure.

3. *Reactions to Penicillin and Other Drugs; Hives; Angioneurotic Edema*
   These maladies are usually self-limited and respond well to the corticosteroids.

4. *Acute Edema of the Larynx*
   Whether the edema is from infection or from trauma, the use of the corticoids may obviate the necessity of doing a tracheotomy.

5. *Critical Illness and Toxicity*
   The corticosteroids can tide the patient over while antibiotic therapy and other supportive measures are carried out.

6. *Idiopathic Midline Granuloma of the Facial Tissue*
   ACTH and the corticosteroids are the most effective treatment in this serious condition.

7. *Erythema Multiforme*
   In many cases, lesions in the mouth are the first sign of this disease. Hence, the otolaryngologist may be the first to see the patient or may be called in consultation. Corticosteroids with appropriate antibiotic therapy give relief and shorten the course of the disease.

8. *Pemphigus*
   Like erythema multiforme, the first sign of pemphigus is often lesions in the mouth. Until the era of corticosteroids and corticotropin, this disease had been considered fatal. The author has been consulted in three cases of pemphigus. All were treated with corticotropin, corticosteroids and antibiotics. Two are now apparently well on no therapy, and one is free of symptoms on a maintenance dose of hydrocortisone.

9. *Histamine Cephalalgia*
   If histamine and other vasodilating types of therapy fail to give relief, corticosteroids, and particularly intravenous corticotropin, will often bring about improvement. However, vasodilating therapy should be continued.

10. *Contact Ulcer of the Larynx*
    Cauterization or fulguration of the lesion followed by a short course of corticosteroid therapy is well justified in this condition. Granulation tissue is diminished and epithelium can more readily cover the ulcer.

11. *Laryngeal Polyps and Extensive Nasal Polyps*
    The following regimen is recommended in these conditions: (a) surgical removal of the polyps; (b) a short course of postoperative corticosteroid therapy; (c) elimination of etiological factors; and (d) treatment of allergy.

12. *Fresh Stricture of the Esophagus*
    Less scarring results if corticosteroid therapy is initiated immediately. Standard treatment should also be undertaken.

13. *Eczema and Dermatitis of the External Canal*
    Local use of hydrocortisone with the appropriate antibiotic is very effective in these cases.

14. *Bell's Palsy*
    In early cases of Bell's palsy, we have had very good results with the use of vasodilators combined with corticosteroids and a low-salt diet.

15. *Ménière's Disease*
    We are now treating Ménière's disease in a manner similar to that used for Bell's palsy; that is, a combination of vasodilatation, corticosteroids and low-salt diet. The author has not had sufficient experience to comment accurately upon the effectiveness of this form of treatment in Ménière's disease.

16. *Depressed Adrenal Cortices, Preoperatively and Postoperatively*
    This has already been discussed.

17. *Respiratory Allergy*
    Local spray and mist hydrocortisone therapy is not especially recommended. We have had only moderate success with this local type of therapy.

18. *Postsurgical Edema*
    In surgical cases in which postoperative edema is often a problem, corticosteroids can be effectively used. The author has found these

hormones especially useful following nasal reconstructive surgery.

19. *Status Lymphaticus*
This condition is supposedly due to a hypoactivity of the adrenal cortices and may result in death from very mild stress. Death from anesthesia furnishes the most typical example. If intravenous hydrocortisone could be administered early enough, recovery could be anticipated.

20. *Anaphylactic Shock and Other Forms of Shock*
Intravenous hydrocortisone is one of the most effective measures.

21. *Emergency Surgery on Debilitated Patients*
When it becomes necessary to do surgery on debilitated patients, stress hormones should be administered just before and a few days following surgery.

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MEDICAL ASPECTS OF HYPERPLASTIC SINUSITIS

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HYPERPLASTIC sinusitis may be defined as a type of chronic sinus disease manifest by polypoid thickening of the nasal and sinus mucosa (figs. 1 and 2).

About two years ago I prepared a paper[1] on this subject in which I stated that I thought this disease was due to local sensitization of the submucosal tissues to bacterial proteins, most often those of the Gram-positive cocci. I think that the observations of A. R. Rich[6] are pertinent, and I quoted him as follows:

> The *tuberculin type* of hypersensitivity can play an important role in the pathogenesis of infection. As a result of the development of this type of sensitization, even micro-organisms which possess little toxicity for the normal body become able to damage seriously the tissues of the sensitized one.

My reasons for believing that hyperplastic sinusitis is due to bacterial allergy follow:

1. Unilateral cases, 24 per cent
2. History of onset with acute respiratory infection
3. Rapid improvement with penicillin, if the germs are sensitive to it
4. Increase in symptoms produced by large doses of vaccine

I still think this thesis is true, but during the last two years I have been asking myself what derangement of physiology *permits* the tissues to become sensitized. The clinical observation that cortisone taken by mouth causes rapid shrinkage of this hyperplastic tissue, our histologic examinations showing dense infiltration with lymphocytes and plasma cells, and Abraham White's report[12] that corticosteroids cause rapid dissolution of lymphocytes have made me think that relative deficiency of glucocorticoids might be a predisposing factor.

I recently reviewed the charts of another series of 50 patients to see what I could learn about the subject. These patients were observed for at least twelve months and the cases were carefully studied. Eighty-six per cent of the patients were over 30 years of age and the largest number of patients were in the sixth decade of life (table I). Twenty-seven patients were males, and 23 were females; this is too small a sex difference to indicate a gonadal deficiency. Six of the cases were unilateral.

TABLE I

AGE RANGE AT TIME OF TREATMENT OF 50 PATIENTS HAVING CHRONIC HYPERPLASTIC SINUSITIS

AGE IN YEARS	NO. OF PATIENTS
1-10	0
11-20	0
21-30	7
31-40	9
41-50	13
51-60	15
61-70	4
71-80	2

I am led by these findings to believe that there may be an exhaustion or deficiency of some physiologic process which *permits* the sensitization to de-

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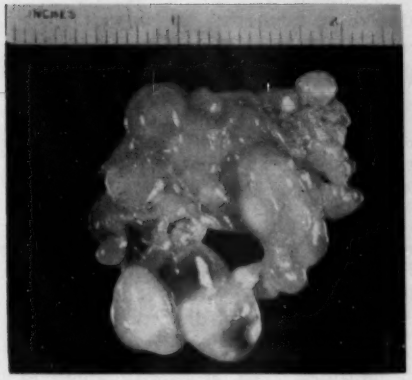

FIG. 1—Lining of an antrum from a patient with long-standing sinusitis and asthma. Note polyps and discrete intramucosal abscesses. Several of the larger abscesses were ruptured in the process of removing the lining.

autonomic reactions are endocrine-mediated. Some patients probably inherit a poor endocrine system which is inadequate to meet the stresses of ordinary existence. As Hilger[3] said: "Outspoken gross signs of endocrine imbalance are the exception, not the rule." The spectacular response of this group of patients to treatment with ACTH or to one of the glucocorticoids makes it easy to believe that chronic pituitary or adrenocortical deficiency may be a factor permitting development of this and possibly other allergic phenomena. It seems that in hyperplastic sinusitis, as in some other hyperergic states, the body suffers more from exaggeration of its

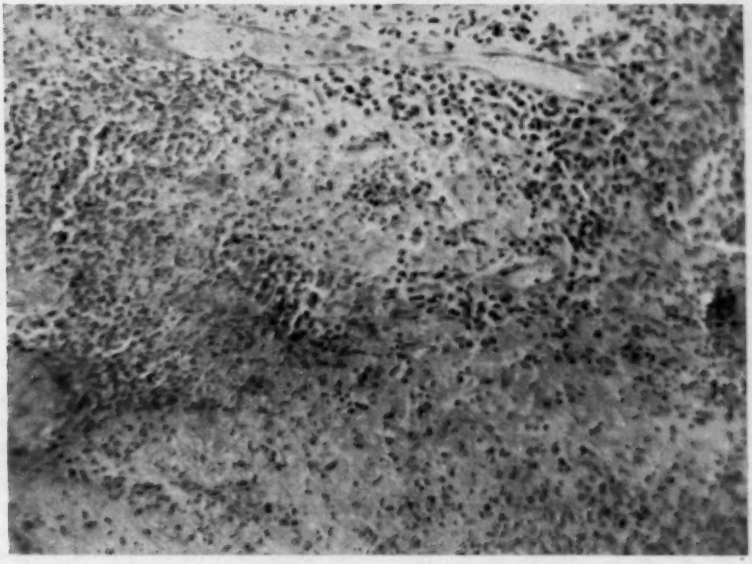

FIG. 2—Histologic appearance showing dense infiltration with plasma cells, lymphocytes and eosinophils. In many areas, plasma cells and lymphocytes greatly outnumber the eosinophils. The areas undergoing necrosis form the chronic abscesses. (Courtesy of Dr. Thomas Hepler)

velop. H. L. Williams[15] suggests that autonomic dysfunction explains many clinically observed allergic phenomena. I agree, but hasten to add that many own reactions than from the pathogen.

Williams[14] and Kahn[4] suggest that "allergy" may be an instance of hyperimmunity.

TABLE II

TYPES OF ALLERGIC SENSITIVITY PRESENT IN 50 CASES OF CHRONIC HYPERPLASTIC AND SUPPURATIVE ETHMOID SINUSITIS

SENSITIVITY	NO. OF CASES
Bacterial allergy	50
Inhalant sensitivity	17
Emotional sensitivity	16
Food sensitivity	9
Physical allergy (cold etc.)	7
Drug sensitivity	6
Endocrine defect	?

Table II summarizes some of the data for my series of 50 cases. After all possible factors had been considered and treated, and after the infections had been treated to the best of my ability by operation and antibiotics, there were 11 patients who seemed to require maintenance endocrine therapy with either ACTH or one of the glucocorticoids. With it they remained symptom free. Without it, they had frequent recurrence of infections, rapid regrowth of polypoid tissue in the ethmoid areas, and recurrence of granular tissue on the upper third of the septum. All of these 11 had asthma as well as hyperplastic sinusitis. Twenty-nine of the 50 were asthmatic, but only 11 of the 29 required maintenance endocrine therapy. The maintenance therapy was used primarily for relief of asthma, but striking additional benefits observed were the complete freedom from nasal symptoms, the absence of regrowth of polypoid tissue in the ethmoid areas, and absence of regrowth of granular tissue on the upper half of the septum.

The following case report will serve to indicate why I think an endocrine insufficiency may be the basic defect in physiology which *permits* the development of at least this type of allergic disease. It also serves to show the limitation of numerous methods of treatment which were utilized with but temporary amelioration of symptoms.

The patient, J. S., was first examined by me in 1942, 13 years ago, when she was 14 years of age. Chief complaint, complete nasal obstruction on the left side of four months' duration. First operation, August 1942, was for removal of nasal polyps and left intranasal ethmoidectomy. Second operation, September 1949, was a bilateral intranasal ethmoidectomy, removal of polyps and submucous resection of the septum. At that time the patient also had granular hypertrophies on the *lateral wall* of the nose on each side in front of each middle turbinate. These areas were treated by electrocoagulation. All skin tests were negative except for a weak reaction to house dust extract. My deduction was that her trouble was entirely due to bacterial allergy. Third operation, May 1950, was a bilateral intranasal ethmoidectomy and removal of nasal polyps. Three ethmoidectomies to remove regrowth of polypoid tissue should be enough to make anyone realize that he was not getting at the real cause of the disease.

The patient's fourth admission, in August 1950, was for treatment of severe asthma. Previously she had had severe asthma every time she had had a head cold, and this also suggested bacterial allergy. Her brother had polyps, and her mother had asthma which was relieved during pregnancy. This suggested poor endocrine inheritance.

She was admitted for the fifth time in November 1950. The chief complaints were nasal obstruction, yellow nasal discharge, cough and asthma. Chest roentgenograms were typical of tuberculosis, but repeated sputum examinations were negative for the presence of acid-fast bacilli, and smears showed many eosinophils. Treatment with penicillin plus 75 mg. of cortisone daily, produced rapid clearing of all nasal symptoms and complete disappearance of the pulmonary densities, thus indicating Loeffler's syndrome.

The patient had six more hospital admissions during the next three years, for treatment of suppurative rhinitis, bronchitis and asthma. Each time, all nasal and bronchial symptoms cleared up within a week in response to cortisone with or *without* penicillin. Each time, cortisone, 75 mg. daily, caused disappearance of polypoid tissue in the ethmoid areas and the red granular hypertrophies on the upper half of the septum and return of sense of smell. She had a healthy baby in December 1953. She is now completely free of nasal and

chest symptoms, and her nasal mucosa looks normal. She is taking 10 mg. of prednisone and two grains of thyroid daily.

The need for exogenous thyroid by patients using maintenance glucocorticoid therapy is shown in the article by Wolfson and associates titled "Corticogenic Hypothyroidism," which appeared in the Proceedings of the 2nd ACTH Conference.[16] They state, "Corticogenic hypothyroidism may cause a serious unresponsiveness to ACTH or cortisone which persists until the thyroid deficiency is corrected."

COMMENT

This case report shows the futility of relying solely on sinus surgery for the treatment of these patients when they respond so well to endocrine therapy. It also shows that maintenance therapy with one of the corticosteroids does not interfere with normal pregnancy and gestation. In fact this patient had never been pregnant until she started taking cortisone. There is nothing in the history to suggest food or inhalant sensitivity, and a trial with autogenous vaccine was without benefit.

For this particular patient, effective maintenance doses appeared to be 50 mg. of cortisone, 40 mg. of hydrocortisone, or 10 mg. of prednisone plus thyroid. This case report makes it seem obvious that if we are to treat this type of patient properly and effectively, we must consider endocrine factors.

Time will not permit listing the many other case reports which parallel the one just given in such detail. Thirty-eight of the 50 patients had endocrine therapy for varying lengths of time without development of undesirable reactions.

During *acute* exacerbations of infection and during the postoperative period it is wise to administer antibiotics if glucocorticoids are being used, because there is some evidence that they depress immunity. Forty-seven of the 50 patients had some type of antibiotic.

There are several theories which might be used to explain the clinically observed phenomena resulting from administration of ACTH or one of the glucocorticoids. Selye's book entitled Stress[7] and the four Annual Reports on Stress[8,9,10,11] furnish much food for thought.

Abraham White[13] said:

> ACTH is secreted in greater than normal quantity following exposure of the organism to a wide variety of unrelated stimuli: inanition, alterations in environmental factors, for example cold, heat and anoxia, trauma in the form of tissue injury, fracture and hemorrhage, injections of bacterial toxins, toxic chemicals of the type of arsenic or benzene, anesthetics such as nembutal, foreign proteins and a variety of substances normally produced in the organism, for example histamine, epinephrine, thyroxin and estrogens. The increased release of adrenal cortical steroid hormone as a consequence of any of these stimuli does not occur in the absence of the pituitary and each of the stimuli therefore has a common physiological basis, i.e. its capacity to increase the output of pituitary adrenocorticotrophic hormone. Thus, an increased secretion of epinephrine in the normal individual will elevate the rate of release of pituitary ACTH and result in an increased rate of secretion of the hormones of the adrenal cortex. . . . In addition it has been demonstrated recently that adrenal corticosteroids contribute to certain aspects of immune cellular responses including development of phagocytic cells, the production of immune globulin and an increased resistance of tissue to the penetration of foreign substances.

Hansel[2] suggests that staphylococcus toxoid may be effective by increasing the secretion of ACTH. The literature of the past five years makes it increasingly clear that the therapeutic effect derived from the use of such substances as histamine vaccines or staphylococcus toxoid may be due to increased formation of ACTH.

I don't know whether the clinically

observed improvement resulting from glucocorticoid therapy is due to neutralization of hyaluronidase as suggested by Rawlins[5] or to the increase in immunity mechanisms described by Abraham White, but I do know that clinical results justify their use.

CONCLUSIONS

1. Hyperplastic sinusitis is due to bacterial allergy.
2. Subclinical endocrine deficiency may be a predisposing factor permitting the development of bacterial allergy.
3. Coexisting food and inhalant sensitivities may be aggravating factors, but their management does not cure well-developed cases.
4. Vaccine therapy is of benefit only in early cases.
5. Surgical treatment is necessary for advanced cases.
6. Postoperative use of antibiotics hastens healing.
7. Postoperative use of glucocorticoids causes smoother and more rapid healing.
8. Maintenance endocrine therapy may be necessary.

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FOOD ALLERGY

Clinical Features of Food Sensitization

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IN this presentation there will be brief discussion of a number of features of food sensitization which should be common knowledge for every practitioner of medicine but which are particularly pertinent to the practice of otolaryngology. There is definite need for close observation. This is not a gift but is the result of systematic study of a patient before and after specific contact.

I should also like to stress the importance of making a specific diagnosis before assigning the term psychosomatic to the allergic manifestation. It is paramount that one approach this problem guided by two basic requirements: first, the ability and willingness of the diagnostician to cover specific etiologic contacts fully, and second, study of the case over a sufficiently long period of time that an accurate, clinical concept is obtained.

THE DIAGNOSIS OF THE FOOD SENSITIVE INDIVIDUAL

The first step, obviously, is that of history taking. This should bring out contacts, particularly in terms of specific inhalants, and should include a survey of the eating habits of the patient. Any attempt to use history taking as an accurate means of diagnosis of food allergy is filled with potential errors. You do not know what you ate yesterday; neither does the patient know what he ate. Furthermore, in modern-day living, foods which seem to a patient to be one item may combine many products. One example will suffice: if a patient eats pork sausage cured with one of the better-known pepper mixes, he does not eat pork alone but he does eat a total of twenty-three different foods. Furthermore, memory being what it is, it will be found that a continuation of the history, particularly by use of the alphabetical food list, on which the patient is asked to check every day for a week, the days each food is used, will prove to be far more exact. In this regard keep in mind that most of your food-sensitive patients will have symptoms from those foods which are included in practically everyone's diet unless for specific religious or concomitant disease syndromes, some of these common ones are eliminated. Such foods will include wheat, corn, eggs, milk, Irish potatoes, beef, and pork. One could almost add citrus fruits to this group. It is extremely important to keep in mind that sensitization is apt to occur to those foods which constitute the common diet. There are, of course, sensitizations to rare foods, but these are frequently recognized by the patient. The sensitizations to foods which are used three or four times a week are the ones requiring careful diagnostic procedures.

Evidences of food sensitization in the history are rather limited in terms of identifying a specific food. It has been a fairly constant finding that if a patient complains of abdominal distress from excessive intake of cooked or partially baked wheat products, he is wheat sensi-

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tive. But, by and large we have found that a patient's idea of which common foods are the cause of his symptoms is just as apt to be false as true. Nothing takes the place of specific diagnosis, supervised by the physician. The history will be more apt to reveal the presence of food allergy in general than to determine a specific causative agent. An abrupt increase of symptoms following the meal time, periodic attacks at 11:00 in the morning or at 4:30 in the afternoon, may be indicative of food sensitivity. Also, the acute exacerbations which occur sometimes in the middle of the night, although the patient lives in the same house, sleeps in the same bed, and works at the same job as always, are suggestive of a food allergy.

Three criteria are highly suggestive of food sensitization: (1) itching of the nose and the roof of the mouth, (2) typical acute exacerbations which last from eighteen to seventy-two hours, and (3) typical exacerbations which waken the patient out of a sound sleep. If two of these are present and the patient has not responded satisfactorily to inhalant therapy, food studies should be carried out.

THE TERMINOLOGY

In order that we may fully understand each other, a discussion of the clinical implication of various terms is in order. We define food allergy as a reaction to a food which produces a definite series of clinical symptoms under correct conditions of testing. This reaction should be specific. It should be reproducible at will; in other words, it should occur consistently under correct conditions of testing. You will note that this definition does not include the stipulation that there should be a positive skin test. When you make a scratch test or perform an intracutaneous test with a food extract and obtain a reaction, you can only state to the patient, "You have given a skin reaction to a food." It is not correct to tell the patient that he is sensitive to the foods simply because they produced skin reactions. It has been our observation that for every twelve positive skin tests to wheat, there were only five patients who actually had allergic symptoms from eating the wheat; therefore, there were seven who had no allergic symptoms and yet gave a distinct skin reaction. Keep this point in mind; it is easy to see that if one considered sensitization as being identical to skin reactions, various desensitizing methods would work. As a matter of fact, there would be seven chances to five for success. Thus, one could always improve the state of health of the majority of his patients by desensitizing them to this-or-that extract of wheat, egg or milk.

Our next point of interest is the types of food sensitization. Food allergy can be considered as being either fixed (permanent) or cyclic (varying from time to time). A fixed food allergy is not alleviated or cured by the omission of foods. Cyclic food allergy is the more common, and in this type of allergy, usage tends toward sensitization and omission tends toward tolerance. It would appear that approximately 95 per cent of food allergies may be classified as cyclic. There is one interesting phenomenon in food symptomatology which accounts for the fact that many men trained in the subject of allergy fail to recognize food sensitivity in their patients; that is the phenomenon of masking. This implies a reduction or a complete disappearance of symptoms when the patient eats the very food to which he is sensitive. This improvement, or eradication of symptoms, will last from an hour up to three or four hours. I have reported the case of a milk-sensitive individual who had five major abdominal operations in seven years for his milk allergy. This case was diag-

nosed as peptic ulcer, and the patient was repeatedly put on a therapeutic regimen using the Sippy procedure, with a resultant improvement or control of his symptoms for seven to ten days. Yet milk was the cause of his trouble. Therefore, if you want to learn whether or not a given patient is food sensitive, you should take the food out of his diet for four days and then feed it back to him in its pure form. You do not test wheat by giving crackers to the patient, because crackers contain pork, cottonseed, yeast, milk, and wheat. Wheat should be tested by using cooked, cracked whole wheat. Deliberate ingestive trial feedings should be run the second meal of the day, and the patient should be observed for at least one-half hour before feeding and an hour and a half following the feeding. Should there be no reactions with the initial feeding, it is wise to feed approximately one-fourth the original amount of food and continue observations for thirty minutes following this second feeding. The observer should note what the patient does, such as sneezing, blowing of the nose, coughing, clearing of the throat. These are the objective symptoms to which one more might be added — the development of pallor. This is more apt to occur in children than adults. There are subjective symptoms, such as tiredness, chilliness, aches, and headache. There might be occasions when a patient of an otolaryngologist has not only nasal symptoms but also eczema, and in those cases redness and itching of the skin might be observed following the eating of the test food.

There is another term commonly used, "latent sensitization." This can be confusing. It occurs when, either deliberately or by accident, a food has been out of his diet for several months, a patient may eat it and may find that his former sensitization has seemingly disappeared. However, when the food is repeated the next day, or within a short time, the second eating is followed by a reaction. This "latent" reaction occurs only to articles of the diet which have not been used for a period of time, usually a matter of weeks but in rare instances as brief a period as twelve days. Keep this reaction in mind when adding foods to the diet which have been omitted over a considerable period of time.

Another term frequently used in describing food sensitization is "cumulative reactor." This refers to foods which do not cause symptoms unless they are used for three to seven meals in close succession. They are absolutely asymptomatic if taken once a day, every other day, or once every third day. It is important to make the diagnosis of "cumulative reactions" because we can then increase the number of foods in the patient's diet by interval usage of the foods with the cumulative type of sensitization. There is only a difference in degree between latent sensitization and cumulative reaction.

The next term of importance is "concomitant food sensitization." This implies a reaction to a food in the presence of an inhalant which does not occur when the food is ingested in the absence of the specific airborne product. There is often a concomitant reaction between foods and dust, although those between pollen and foods are more commonly known. It is of great interest to know that when a patient eats a food for which there is a concomitant reaction, it results in an increased sensitization, the endpoint of reaction may shift one dilution (1:5 dilution), necessitating a dropping of the dose during the period of this food reaction. When the effect of the food has disappeared, the dose must again be increased to its previous level. In patients who have a very narrow zone of relief from inhalant treatment, this change of sensitization result-

ing from the use of a concomitant food reactor becomes of great importance. Incidentally, there is an increase of sensitization in the presence of certain specific infections, and in these cases, too, the therapeutic inhalant dose must be temporarily lowered. There are other implications of the concomitant reaction. Suppose you work out a patient's diet in the ragweed season and must eliminate a number of important foods. It should not be assumed that these foods need be omitted after frost. In fact, it is an obligation on the part of the diagnostician to determine whether such foods must continue to be banned after frost. The end of the hay fever season in various parts of the country may differ, but in ours it occurs when the temperature has fallen to 25 degrees Fahrenheit and there has been at least an inch of rain. It is a surprising fact that during the past two years of continued drouth, in a number of instances the inhalant (pollen) doses had to be continued up until early January.

There is another food reaction which we term "synergistic." This is not nearly as common as formerly thought. The effects of two allergenic foods are not added, they are multiplied. It is not 4 plus 4 equals 8; it is 4 times 4 equals 16. Here, again, careful study of these foods individually shows that each alone produces symptoms. However, a superficial history-taking will include the statement of the patient that he has trouble only when the two foods are taken together.

Finally, we come to the type of food sensitization which, above all others, concerns specialists in otolaryngology. This is the thermal type of food sensitization. There may be many other factors involved in this reaction beside allergy, but as long as allergy is a trigger mechanism and its control means relief for the patient, and until such time as there is specific therapy for the thermal mechanism, it is imperative that these patients be studied and diagnosis made specifically. The distinguishing feature of the thermal type of food sensitization is that it produces the characteristically clear nasal secretions of the allergic reaction for approximately forty-eight hours, then the secretion becomes purulent, the temperature rises, and the purulent secretion continues for at least three days. This is true when there has been only one exposure to the antigen; the disorder automatically clears up in five days. Clinically, a most common manifestation of this type of reaction is seen in the patient with asthma who reports that he had a cold and after forty-eight hours it slipped down into his chest and gave him asthma. Actually this is due, in many instances, to a single allergen, either a rare inhalant or a food. Of course I am assuming that such patients have been subjected to a very exacting and accurate diagnosis of inhalant allergy. Therefore, the possibility of an inhalant causing a reaction of this type is materially reduced, yet it may occur in patients who have sensitizations to rare inhalants. It is interesting that the individuals with thermal food sensitizations become acutely sensitive to chilling as a result of eating the allergenic food. They are not aware of the fact that this chilling is the result of an allergic reaction. They are only conscious of their sensitiveness to cold and deliberately protect themselves; and the more they protect themselves from variations in temperature, the more highly sensitive they become to such changes. Therefore, some of these patients may lead almost cloistered lives; they do not engage in any winter activities or normal outdoor exercises. Patients with thermal food allergies may have other types of food sensitization. In one instance a patient was extremely sensitive to wheat. This would result in an antrum infec-

tion if the patient was exposed to a temperature of 35 degrees Fahrenheit. However, at the identical time far more severe nasal allergic symptoms were produced by orange juice; they resulted in the total occlusion of both nares over a period of hours, yet this allergic reaction to orange juice never resulted in purulent secretions and a rise in temperature. There is a difference, therefore, in the type of allergic reaction to certain foods, and this difference is the predisposing factor in the development of infectious processes or so-called infectious processes.

When infection occurs, it should be treated as such and not just as an allergy alone. Conversely, the probability of allergy should be evaluated in many patients with recurrent infections. A few of these patients are allergic and yet fail to react to ragweed, to dust, to Alternaria, or to a combination of these three. Titration tests for sensitivity to these allergens should be performed in keeping with the indicated procedure in order to avoid reactions. As an example of this, I recall seeing a patient in Dr. Hansel's office whose history was that of more or less continuous colds. He had a great deal of purulent nasal secretion, and when taking the history Dr. Hansel felt that "this surely must be an infection." However, he decided "just for fun" to run a nasal smear. The smear contained approximately 98 per cent eosinophils. This experience impressed me with the importance of a complete examination of patients with recurrent colds. One final point of interest: an untreated or incorrectly treated patient sensitive to house dust may also have an acute sensitization to cold.

PENICILLIN REACTIONS

All of you have had experience in the use of penicillin and are acquainted with the occasional occurrence of penicillin reactions. While this is not directly a food problem, it is closely related to certain foods, namely those containing yeast. There is a synergism between various yeast-containing foods and a penicillin sensitization, which, in some instances at least, is the cause of long-continued penicillin urticaria. I can think of no other cause of urticaria which will persist for the length of time that is sometimes observed following a reaction to penicillin. In testing patients for penicillin we make dilutions in the same way as we do with pollen. The testing is started and is continued by the coseasonal technique.[2] The endpoint of reaction is not a wheal with a zone of erythema; the papula is merely pink at the end of ten minutes. Treatment consists of approximately .10 cc. of the endpoint dilution, followed in 48 hours by .20 cc., and followed in 72 hours by .30 cc. In a series of fifteen cases treated in this manner, three of the patients violently ill, complete freedom of symptoms was obtained following the three injections. However, in treating these patients we eliminated all yeast-containing foods, including all fermented drinks, root beer, all cheeses, bread, vinegar, and any of the cereal grain foods enriched with these products. After freedom from symptoms had been obtained for several weeks, the foods have in all cases, in our present experience, been returned to the diet without a flare of symptoms. Not all patients will actually have a synergism between yeast-containing foods and penicillin, but the frequency is high.

Following Hansel's use of TOE in the treatment of patients sensitive to trichophytin, oidiomycin and epidermophytin, we have also found a synergism between yeast-containing foods and the eruption due to these infections. It has been our experience that bread and vinegar are the two most potent synergistic yeast contacts.

FOOD TESTING BY THE OTOLARYNGOLOGIST

An ideal setup in the office would be to have a room set aside for food testing, with a trained technician in attendance. However, this is not always possible. In lieu of this, we have advocated, and a number of otolaryngologists are using, the technique hereafter described.

The suspected foods are eliminated from the diet for four days. The patient eats a normal breakfast and reports to the office after he has been without food for four hours, without water for one hour, and without smoking for two hours. The patient's nose and throat are examined for the presence and amount of secretions and obstruction. The food is then fed and the patient observed so that any coughing, sneezing, or other signs, can be recorded. The nose and throat should be re-examined at the end of forty minutes, and at the end of ninety minutes. It is possible to observe four patients without too much interference with other work, by carrying out this technique. There should be some definite training of the patient to observe and record symptoms. We first teach our patient what to record and then give him instructions for registering symptoms in a printed form. This will prove to be accurate in approximately 85 per cent of the tests. The interest and cooperation of the patient are very important factors in therapy. It is my plan to remove no food from the patient's diet unless we are able to precipitate symptoms. Certainly a food should not be removed solely because a patient "thinks" it bothers him. It should be checked, and if it does not produce symptoms, it should continue in the diet.

Another procedure includes the use of diet charts. But diet charts are not as satisfactory as we would like for them to be; the best they can do is to indicate the variety of foods the patient is using, and the constancy and kind of symptoms which he may have. They may reveal the reactions the patient has at four-day or five-day intervals. Foods taken every other day, or every third day, or daily, may be a cause of symptoms. This cannot be determined by the use of diet charts.

Again, you may wish to use the elimination diet. The best elimination diet is one made up of foods which a patient has not used for six months or longer, plus cane sugar, Crisco or other hydrogenated vegetable oils. I have never seen a proved sensitization to hydrogenated cottonseed oil, although at present a problematical sensitization is under study. I have seen only a few cane-sugar-sensitive patients, so we invariably use cane sugar. Beet sugar is more apt to cause allergic reactions than cane sugar. A patient may be kept on this restricted diet for four or five days, then additions are made to the diet by the escalator system. It is not a wise plan to place patients on a diet of one, two, or three foods. This is due to the fact that when a patient is so ill that it is impossible to make a specific diagnosis without restricting him to one or two foods, he is too ill to be placed on such a restricted diet. This is a matter of pure logic. If a patient has become sensitized to a great number of foods, it is evident that he must sensitize easily. Therefore, the more you restrict the diet, the more new sensitizations you will have to deal with. You will get much better results if you use a widely diversified diet and check the important foods in sequence, one at a time.

One can insist that a food-sensitive patient use the rotary, diversified diet,[1] because this diet will automatically eliminate the latent sensitizations and cumulative reactors. In a great number of instances, the patient will create a satisfactory masking and the severity of

many of his most annoying symptoms will be reduced, or they may cease altogether. However, there is no procedure so satisfactory as the specific and absolute elimination of the foods to which the patient is sensitive, until he gains tolerance. When tolerance is obtained, the patient must be taught how often a food can be used without losing his tolerance. It is necessary to give the patient sensitive to wheat, egg, milk, or corn exact information as to where he will contact these foods. Many patients have no idea what is meant when you tell them to quit eating wheat, eggs, or milk. Invariably there is always a groceryman or a waitress willing to assure a patient about a wheat-free bread when, in reality, there is no such bread. It normally is not possible to purchase rye seed free of wheat, so why would it be possible to obtain rye bread free of wheat?

It is important to realize that cross-sensitization in the cereal grain family is extremely high. Therefore, the foods closely related to wheat, even if not cross-contaminated with wheat, rapidly break down and become active allergens.

It is also important in food diagnosis and therapy to ascertain whether the patient has actually eliminated the food to which he is known to be sensitive. This requires attention and detailed instruction on the part of the physician. Allergic individuals are like diabetic patients. They must be indoctrinated in a whole system of care and knowledge, and a casual resume of the problem will not be effective. Before you decide that the patient has failed to respond to treatment, make sure that he has actually eliminated the foods to which you have proved him to be sensitive.

Another fact of great importance. The earlier in life a patient has become sensitive to some foods, the easier it is for him to become sensitive to others. A five-year-old child with a severe perennial nasal allergy which he has had since he was eight months of age, sensitizes easier than the person who has lived forty years before developing any food allergies. These two individuals must be treated accordingly. It is very necessary in the younger group to spread out the diet, to get away from the constant usage of a limited number of foods. In the patient who develops allergy late in life, absolute elimination with moderate restrictions is frequently a satisfactory procedure.

SUMMARY

In the diagnosis of food allergy there is need for close observation of the patient and exacting specific diagnosis before assuming that the symptoms have a psychosomatic origin.

There is a need for continuous history taking and evaluation of the cause-effect relationship. It is, therefore, important that patients be under observation for at least a twelve-month period.

The physician should realize the limited value of skin tests for foods, should be aware of the different types of food allergy, and should be conversant with the possible responses to the ingestion of foods, particularly the masking effect and the hyperacute reaction when a food is eliminated for a short period of time.

In the practice of otolaryngology there will normally be a great number of patients with thermal types of food reactions.

It is important in the treatment of reactions to penicillin and to TOE to evaluate yeast-containing foods, which may, and frequently do, have a concomitant effect.

Finally, determine whether the patient sensitizes easily or slowly and arrange

a diet which is compatible with the type of this patient's tolerance.

In all cases furnish patients who are sensitive to complex foods, such as wheat, corn, eggs, milk, printed instructions concerning specific attacks. Recheck with the patient and make sure instructions are being followed.

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A CONJUNCTIVAL IRRIGATOR

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THE conjunctival irrigator consists of a specially constructed goose-bill metal tip, two inches long, attached to a three-ounce rubber ear syringe (fig. 1). There is a slit opening in the tip, by which a broad thin stream of fluid is obtained.

The advantage of the instrument is that the tip is easily inserted under the lids so that the cul-de-sac can be thoroughly irrigated.

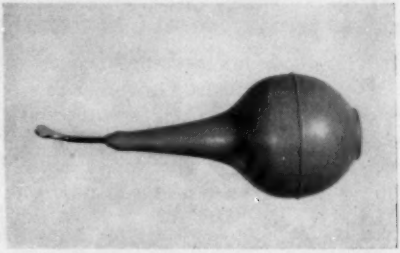

FIGURE 1.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

NEW CLIPS AND CLIP-APPLYING FORCEPS FOR SCLERAL SHORTENING PROCEDURE

RAMÓN CASTROVIEJO, M.D.
NEW YORK, NEW YORK

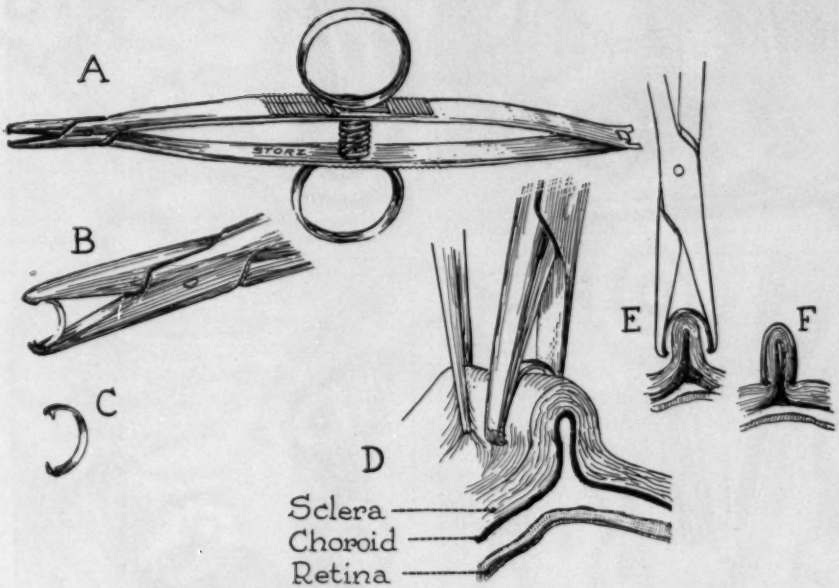

FIG. 1—(A) Clip-applying forceps. (B) Detail of the jaws holding the clip. (C) Monel metal clip. (D,E,F) Application of the clips to the folded sclera.

IN the June 1955 issue of the *Archives of Ophthalmology*, Dr. William G. Everett reported a new scleral shortening operation consisting of folding the sclera outwards and maintaining the fold by mattress sutures. In his article, Dr. Everett states that since the preparation of his paper, he has learned of a similar operation independently developed by me. It is correct that at the same time that Dr. Everett was conducting his experimental and clinical studies, I was also carrying out similar studies on humans. Dr. Everett's operation, if not identical, has all the basic characteristics of mine, evaluation of subretinal fluid, an outward fold taken in the sclera, and maintenance of the fold by mattress sutures.

I would like now to report a further simplification of scleral shortening procedure for the treatment of retinal detachment, using metal clips instead of

From the Department of Ophthalmology of St. Vincent's Hospital, New York, New York University Postgraduate Medical School and New York Eye and Ear Infirmary.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

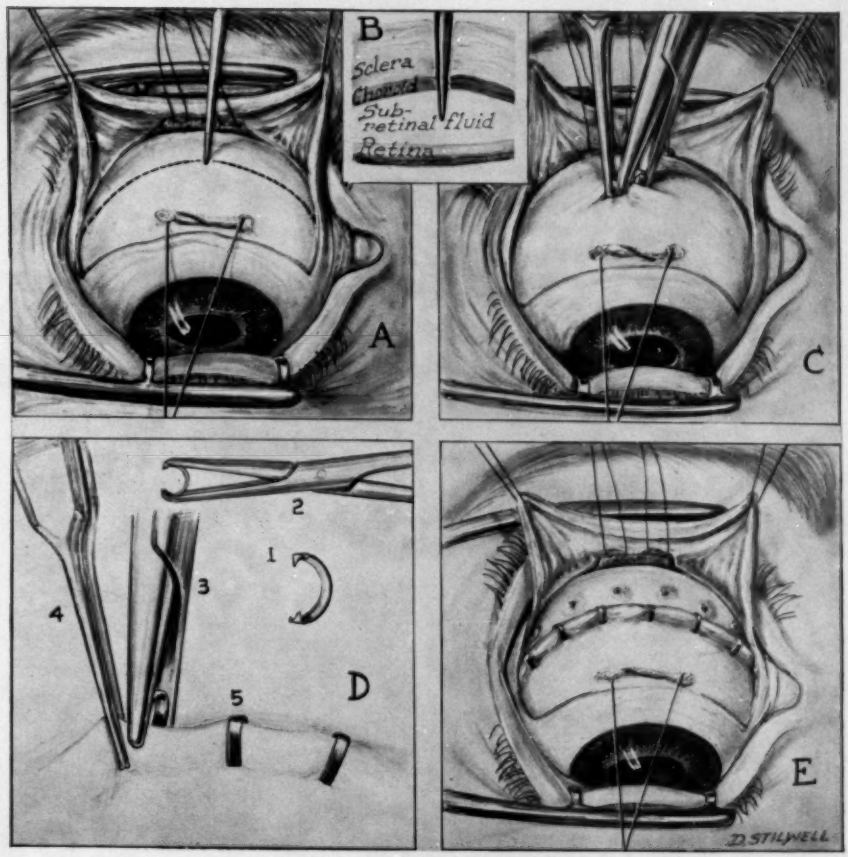

FIG. 2—Different steps of the operation.

sutures to hold the scleral fold. These clips made of Monel metal are 4 mm. long, with teeth measuring 0.25 mm. (fig. 1, C). The clips are held by a clip-applying forceps (fig. 1, A and B) provided with small ridges at the end of the jaws which prevent the clips from slipping. A coil spring provides enough tension to keep the jaws sufficiently taut to hold the clips. Rings for index finger and thumb permit the surgeon to open the forceps to engage the clips. These clips are applied after the subretinal fluid has been drained through a diathermocoagulation puncture, made approximately at the equator, enlarged with a punctum dilator (fig. 2, A and B). The sclera is folded with the aid of fixation forceps. The clip held by the clip-applying forceps is then brought into position and, by closing the forceps, is left securely in place (fig. 1, D, E, F, and fig. 2, C and D). Clips are applied every 7 to 8 mm., and as many as may be necessary are used to complete the desired length of the fold (fig. 2, E). The fold is made along the equator of the eye. After completing the fold, a

fairly normal tension is generally maintained in the globe. About 4 to 5 mm. behind the fold, the sclera is treated with diathermocoagulations and air is injected into the vitreous if an extensive detachment still persists at the completion of the operation.

The metal clips here reported have been used in five cases, most of them very unfavorable, after one or more operations for retinal detachment had failed. The clips seem to be well tolerated. The period of postoperative observation has ranged from two months to over a year. Although it is not possible as yet to appraise the final results of this new approach to scleral shortening because of the small number of cases operated upon and the selection of very unfavorable cases for clinical study, the results so far obtained seem to compare favorably with those of other more complicated scleral shortening procedures. The simplicity of this technique should make it possible for the average ophthalmic surgeon to employ it in many selected cases, and then a more adequate evaluation of the new method may be expected. The clips and the clip-applying forceps are manufactured by Storz Instrument Company, 4570 Audubon Avenue, St. Louis 10, Missouri.

Important Note:

Since the presentation of these new instruments and technique, the author has further observed two of the cases operated upon. Although the retina is still maintained in good position, these cases have developed persistent conjunctivitis accompanied by moderate chemosis and mucopurulent discharge. The nature of this persistent ocular irritation, which has not responded to treatment with antibiotics or cortisone, has not yet been determined. It is possible that the metal clips have caused the inflammatory symptoms by some chemical reaction, or more likely that the clips have acted mechanically, causing irritation either to Tenon's capsule or muscles. At any rate, until further work has demonstrated the innocuity of the clips, the new technique should be considered still in the experimental stage.

NEW MASKS TO LIMIT THE ACTIVE SURFACE OF RADIATION IN BETA RAY APPLICATORS

RAMÓN CASTROVIEJO, M.D.
NEW YORK, NEW YORK

IN order to limit the active surface of radiation of beta ray applicators for treating only the particular zone of the eye requiring the treatment and to protect the surrounding areas, I have devised some masks which fit snugly over the head of the strontium 90 applicator, permitting treatment of circular areas 3 mm. and 5 mm. in diameter, and elongated areas shaped with the same curvature as the limbus, 2 mm. and 3 mm. wide and 8.7 mm. long (fig. 1). These masks of stainless steel, 0.5 mm. in thickness, diminish from 50 per cent to 87 per cent the active surface area of the applicator and reduce proportionately the total dose of radiation received by the eye while confining the radiation to the desired area. They also protect the uninvolved surrounding areas from unnecessary radiation and undesirable postradiation reactions. The importance of reducing the area of treatment as much as possible and diminishing the dosage of radiation received by the eye should be strongly emphasized because, contrary to some reports minimizing the danger of beta radiation, overtreatment may cause very serious complications, including the loss of the eye.

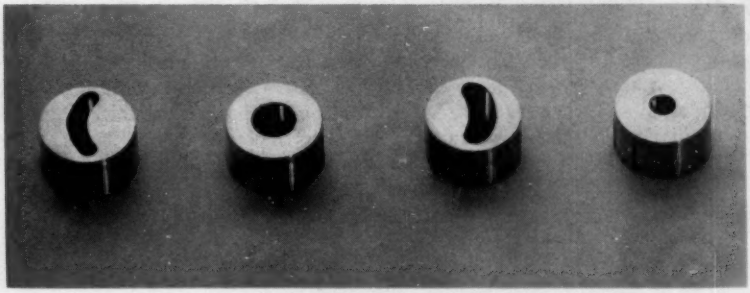

FIG. 1—Screening masks to limit the active surface of radiation of the strontium 90 applicator.

These masks are supplied by Technical Operations, Inc., 6 Schouler Court, Arlington 74, Massachusetts.

From the Department of Ophthalmology of St. Vincent's Hospital, New York, New York University Postgraduate Medical School and New York Eye and Ear Infirmary.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

FLUORESCEIN APPLICATORS: FLUOR-I-STRIPS

SOLOMON S. BRAV, M.D.
PHILADELPHIA, PENNSYLVANIA
and
ROBERT R. FEINSTEIN
UNION CITY, NEW JERSEY
BY INVITATION

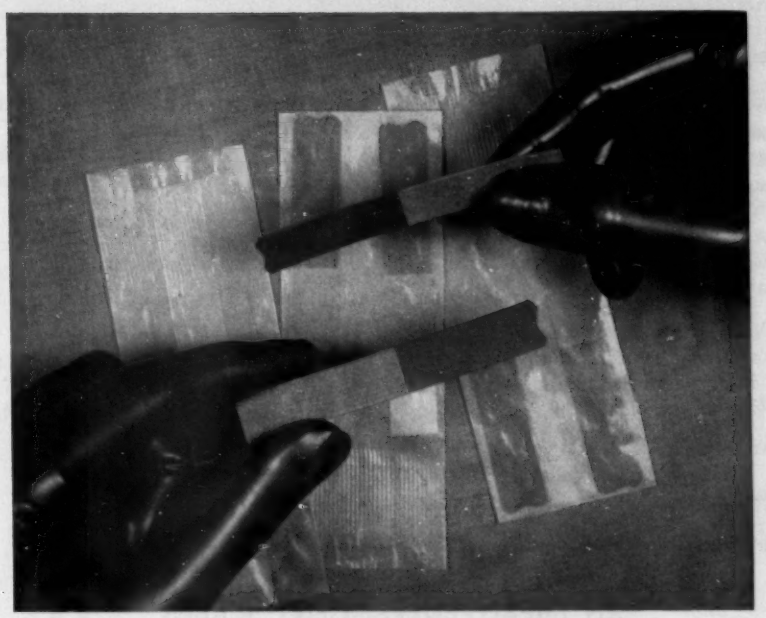

FIGURE 1.

THE use of fluorescein as a diagnostic agent for the delineation of corneal foreign bodies and injuries was introduced by Phlüger in 1882. Other dyes have since been suggested, but because of the sharply defined area obtained with fluorescein, it has remained the stain of choice.

There are a number of objections to fluorescein solution which are well known to all ophthalmologists. The most serious of these concerns sterility of solution. The hazards inherent in contaminated solutions have been reported by many investigators. Theodore and others have shown that fluorescein provides an excellent culture medium for Pseudomonas aeruginosa (B. pyocyaneus). This contamination can occur within a short period of time after

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

a sterile bottle of fluorescein has been opened, due to subsequent handling and excessive exposure.

Kimura's suggestion, that filter papers be impregnated with fluorescein and autoclaved, appeared to us to be a possible solution to this problem. However, ordinary filter paper proved unsatisfactory for this purpose because the lint-free variety is too hard and too stiff. The soft filter papers contain too many fibers protruding from the surface of the paper; these fibers upon impregnation and drying, harden to form minute, needle-like, highly irritative crystals. It was, therefore, necessary to develop a bibulous paper which could be coated with sufficient fluorescein so that when it was applied to the eye, a clinically satisfactory stain of the cornea could be obtained without additional trauma.

We wish to present a new instrument for applying fluorescein to the eye under ideal aseptic conditions without irritation to the globe.

An especially developed sulfite paper, which does not contain the melamine resins customarily used to increase the wet strength of the paper, is coated with a known concentration of fluorescein sodium paste into which a spreading factor has been incorporated. These strips of paper have been laminated between waterproof impermeable grips at one end to enable the ophthalmologist to handle the strips without contamination. These Fluor-I-Strips are obtainable in glassine envelopes which have been heat-sealed and sterilized (fig. 1).*

At the time of application, the curved end of the strip is inserted into the lower cul-de-sac over the punctal area and the patient is instructed to close the lids. Satisfactory staining is obtained within two to ten seconds. No irrigation is required.

The problem of Bell's phenomenon is obviated, since the incorporated spreading agent provides for sufficient diffusion of the stain.

Fluor-I-Strips can also be utilized for the Schirmer test, providing a satisfactory measurement of tear function while simultaneously staining the cornea.

*These fluorescein applicators are obtainable from Ophthalmos, Inc., Union City, N. J.

AN IMPROVED TANGENT SCREEN

RALPH D. GUNKEL, O.D.
BETHESDA, MARYLAND

BY INVITATION

THIS tangent screen is of unique design and incorporates the following features:

1. Standard 4-foot-square black fabric screen with central fixation point.
2. Distance of 1 or 2 meters from head rest, as desired.
3. Noiseless test objects of any desired size and color. These are manipulated, without a wand, by a magnet hidden behind the screen. The operating lever is at a suitable distance to the side of the screen to prevent distraction of the patient.
4. Automatic recording on a conven-

From Ophthalmology Branch, NINDB, National Institutes of Health.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

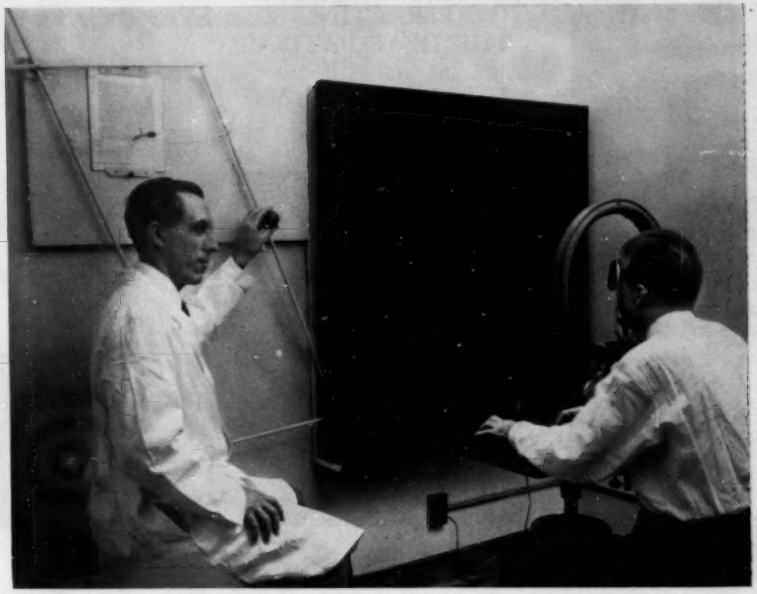

FIGURE 1.

tional type of field chart by a simple mechanical device. A pantograph directs the recording device accurately.

5. An adjustable occluder is placed on the otherwise conventional head rest.
6. The need for pins, tacks, wand, chalk, pencils, grid-projector and other accessories is eliminated.
7. The operator is in a position to observe the patient's gaze at all times, without moving into the patient's field of vision.
8. A circular fluorescent light about the head rest, giving uniform illumination of desired intensity, is optional equipment with this screen.

This improved tangent screen increases accuracy in three ways:

a. Remote control removes the following distracting factors: wand, movement of arm in visual field, use of pins, tacks or chalk to mark isopters, and most of the noises common to other tangent screens.

b. Reduction of examination time to one-half reduces fatigue of patient and examiner.

c. Mechanical recording eliminates inaccuracies of transferring record from tangent screen to paper by pencil.

All good features of a standard size tangent screen at 1 or 2 meters distance are preserved.

The device is simple in construction and therefore could be manufactured and sold at a nominal price.

AN AID TO REFRACTING THE EYES OF THE DEAF PATIENT

JAMES D. MORRISON, M.D.
BILLINGS, MONTANA

THE ophthalmologist often finds himself handicapped when called upon to refract the eyes of the deaf patient, either because the patient has not been fitted for a hearing aid, or has left the hearing aid at home, or has a nonfunctioning hearing aid. The difficulty is too often determined after the patient is in the chair awaiting a refraction.

The examiner is advised to bring an audiometer into the room to aid him in his examination. This can be done if the doctor has an audiometer at his disposal. The use of an audiometer, while better than nothing, has several disadvantages: (1) it is a large heavy instrument, often kept in another room and easily maladjusted by movement; (2) the setting up takes time; (3) most instruments cannot amplify sound into both ears at the same time; and (4) the microphone has to be held with one hand; when it is laid down on the examining table, it causes unpleasant sounds in the patient's ear.

I have frequently found myself handicapped in my attempt to examine the deaf patient. At meetings, I have asked the representatives of various hearing aid companies if an instrument for this purpose is made, but have always been asked, "Why not fit the patient with our hearing aid?" As a result, I have assembled the following apparatus for my convenience: (1) a microphone held in place by a wire around my neck; (2) an amplifier and rectifier connected with the 110 volt alternating current; (3) headphones for both ears. The amplifier unit can be adjusted for volume input from the microphone, frequency of tone and volume of tone.

FIGURE 1.

FIGURE 2.

The amplifier unit is on the back of my examining table, out of the way, and when not in use, the microphone and the headphones hang on a convenient hook (fig. 1). While using this apparatus I have both hands free (fig. 2).

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

A NEW MODIFIED LENS CAPSULE FORCEPS

RICHARD E. LEIGH, JR., M.D.
HOUSTON, TEXAS

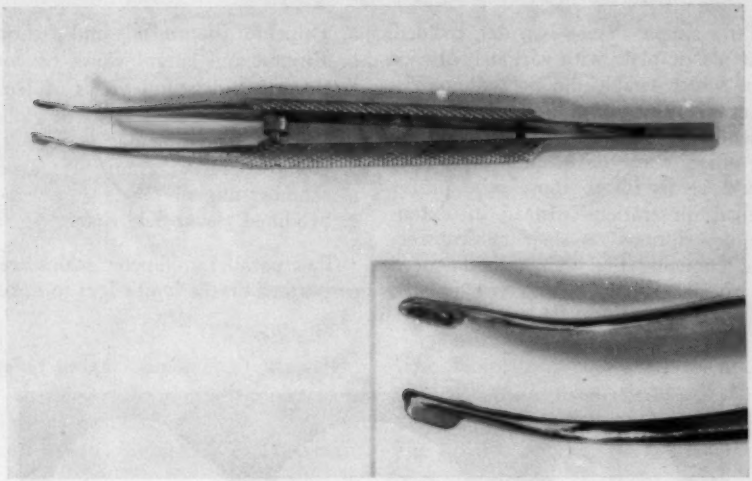

FIGURE 1.

OPHTHALMIC surgeons who deliver the lens with a twelve o'clock grasp have long been troubled by the iris when performing a round pupil extraction. The modified lens capsule forceps was devised to reduce iris trauma to the lowest possible degree and to facilitate the securing of an adequate grasp of the upper pole of the lens.*

A capsule forceps with Arruga jaws was modified by forming a lip on the left blade (fig. 1). The lip extends the length of the jaw and is 1 mm. in width. The lower quarter of the lip is flat, but the upper quarter is sharply beveled. The beveled upper surface of the lip allows the iris to slip freely from the lip as delivery of the lens proceeds.

The normal technique of lens delivery from above needs to be modified only in the following respects: The lip of the left blade is placed under the iris border, which is gently retracted to expose the upper pole of the lens. When an adequate grasp of the lens has been accomplished, an anterior and caudal motion of the capsule forceps is carried out. With this maneuver, the iris quickly slips off the forcep lip, and the remainder of the cataract extraction is carried out in the normal manner.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

*Instrument made for author by Storz Instrument Company.

AN EYE TEMPLATE

MAURICE D. PEARLMAN, M.D.
CHICAGO, ILLINOIS

THIS simple device is a flat, molded, clear plastic plate with variously-shaped holes which enable the ophthalmologist to trace outlines of the eye or its component parts quickly and uniformly on any office, clinic or hospital record. It should be useful to those who prefer labelled illustrations instead of extensive descriptions on their clinical records. In using this template, the need for special rubber stamps or printed forms is obviated; only a pencil or pen is necessary to trace outlines of any of the following:

a. Palpebral fissure, lids and caruncle
b. Frontal and lateral views of cornea
c. Frontal and lateral views of lens
d. Frontal and lateral views of globe
e. Fundus, including disc
f. Gonioscopic diagram of anterior chamber angle
g. Modified visual field chart

Two parallel millimeter scales are incorporated on the long edges to measure lesions, pupils, palpebral fissures, interpupillary distances, etc., and also for exophthalmometry. This last measurement is made by placing one of the specially notched corners against the lateral orbital rim (as is done with the

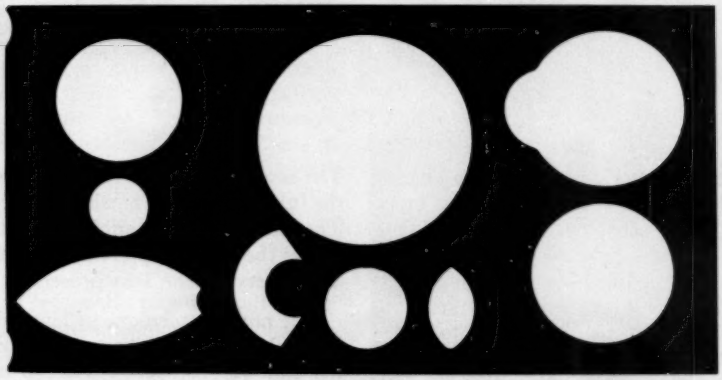

FIG. 1—Pearlman eye template. The two parallel millimeter scales on the long edges are not shown in this picture.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

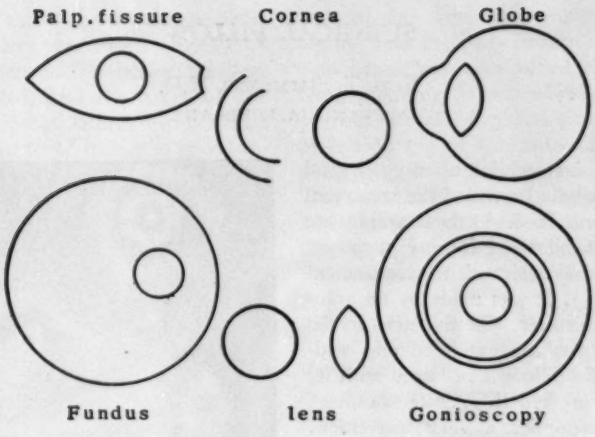

FIG. 2—Sample tracings made with the eye template.

Luedde exophthalmometer) and sighting obliquely for the reflected image of the corneal surface on the tops of the two scales to eliminate parallactic error. The template is designed to fit conveniently in a coat or shirt pocket.

This eye template is distributed by the House of Vision, Inc., Chicago.

SURGICAL PILLOW

NOEL T. SIMMONDS, M.D.
ALEXANDRIA, LOUISIANA

ON the first model of my surgical chair, the original arms of the treatment chair were retained. As these arms were too narrow and short for my purposes, a pillow was designed to supplement them (fig. 1). It was made by an automobile upholsterer. On the new model chair the arms are extended and widened, and the pillow is not used with it; but if one prefers to operate standing, the pillow supplies a very convenient rest for the hands. The pillow has the following dimensions: 22 inches wide, 8 inches deep and 5 inches high. The sides for hand rests are 5 inches wide. The head space is 10 inches wide, allowing space for draping. Three or four folded towels are placed in the pillow under the head to raise the patient's eye to the proper level.

FIG. 1—Surgical pillow. Three or four folded towels may be used to raise the eye to the proper level.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

SURGICAL CHAIR

NOEL T. SIMMONDS, M.D.
ALEXANDRIA, LOUISIANA

FOLLOWING the Sixteenth International Congress of Ophthalmology in London in 1950, it was my pleasure to visit the Barraquer Clinic at Barcelona. I was quite impressed by the chair that Dr. Barraquer sat in to do his surgery and was disappointed to learn that it was made in their own machine shop and could not be purchased on the market. I later visited Dr. Watson Galey's clinic at Bloomington, Illinois, and saw his operating stool and special table. Both of these setups required specially built operating tables, as both the Barraquer chair and the Galey stool were of standard chair height.

To improvise a surgical chair which could be used with a standard operating table, an ordinary specialist's treatment chair was modified by a local machine shop as follows: The legs were made twelve inches longer, and a secondary base having four rollers which work on

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

a lever which raises and lowers the chair off of the rollers was added (fig. 1). A footrest sixteen inches below the seat was also added, and the arms of the chair were widened laterally to 6 inches and lengthened to 17 inches to give support to the hands (fig. 2). The anterior ends of the arms were elevated 1 inch higher than the posterior ends. A diagonal brace was also added to the arm rest, extending 10 inches below it. Three or four layers of cotton padding were placed on top of the arms and held in place with roller bandage and stockinet. The seat can be cut out slightly on each side like a bicycle seat, if the surgeon's thighs are very large. A sponge rubber pad lessens discomfort and fatigue.

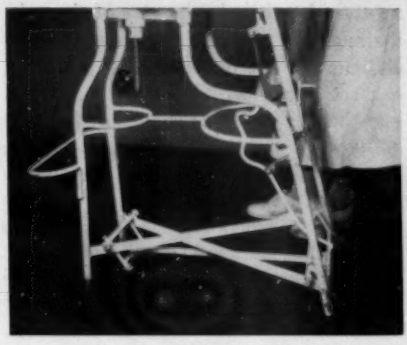

FIG. 1—Surgical chair. Rollers tilted; chair resting on legs.

The patient's head is placed as close to the head of the table as possible, and the head of the table may be elevated one or two notches, depending on the patient's posture. A small sandbag is placed on each side of the head but usually no pillow is used. The surgeon sits in the chair and adjusts the table height before scrubbing. After the patient is prepared and the surgeon has scrubbed, sterile pillow cases are slipped around the arms of the chair. The sur-

FIG. 2—Surgical chair. The new arm, widened laterally, as compared with the original arm.

FIG. 3—Surgical chair in use. Note arm and hand at rest.

geon then sits in the chair and the circulating nurse pushes the chair, with the surgeon in it, up to the proper position to operate (fig. 3): slightly to the right of center for the right eye, and center or slightly to the left of center for the left eye, for a right-handed operator. The chair is then let down off the rollers for absolute stability.

It is my opinion that the use of this chair will give the occasional operator confidence and skill approaching that of the free-handed artist. For those who do a great deal of surgery, it eliminates enough stress and strain to justify its use; I have done as many as seven operations in one day with remarkably little fatigue.

A head light was also copied from one used by Dr. Barraquer (fig. 3). I find it most satisfactory for use during all operative procedures except detachments, when it is in the way of the ophthalmoscope, and capsulotomies or discissions of capsular cataracts, when I can see better with the light projected into the eye from one side.

NEW INSTRUMENTS

If you have a new instrument which you wish to report at the annual meeting of the Academy in October, please notify the Secretary in charge of your Scientific Section as soon as possible. The deadline is September 15. It will not be possible, as formerly, to make arrangements during the meeting for such presentations.

The Secretary for Ophthalmology is Dr. Kenneth L. Roper, 55 East Washington Street, Chicago 2. Dr. Lawrence R. Boies, E537 University Hospital, Minneapolis 14, is Secretary for Otolaryngology.

BRONCHOSCOPIC BIOPSY SPOON

G. ZAKHARY, M.B., CH.B.
CAIRO, EGYPT
BY INVITATION

I UNDERTOOK to design this new surgical bronchoscopic biopsy spoon (fig. 1) because of the high percentage of negative results when ordinary biopsy forceps are used, especially in cases in which the growth is in one of the upper lobe bronchi.

The handle of the instrument forms a vertical limb about 10 cm. long, sufficient for a gentle hand grip. At one end of the handle there is an insulated socket into which the plug of the diathermy cord can be easily fitted if the instrument is going to be used for cautery. During biopsy the cord should be removed. The other end of the handle joins the horizontal limb. It is narrowed on both sides so that it does not obstruct the vision of the surgeon.

The horizontal limb is about 50 cm. in length (a few centimeters longer than the adult bronchoscope or esophagoscope) and about 3 mm. in diameter. It is semiflexible, which serves to diminish trauma to the mucous membrane of the bronchi during surgical manipulations. At its free end there is a socket about 1 cm. deep into which the cutting or cauterizing ends can be screwed for a length not less than 8 mm. Due to this mechanism there is no danger of any of these ends slipping off inside the bronchus or esophagus.

The cutting ends are 2 to 3 cm. long and are formed of three parts: (1) the screw end; (2) the central part, which is about 2 cm. long and is made of spiral malleable metal with a spring action; and (3) the cutting end, made of stainless steel. The cutting end which I use most often (fig. 2) has a champagne-cup shape, with a sharp castellated edge. The cup holds the tissue after it has been cut. Other ends which have been designed are a cutting fork end, a biangular cutting end with a hook, and straight, curved, rounded and oval curets with sharp or blunt edges.

The whole handle and the cutting end work with a lever action with a slight rotatory movement.

This bronchoscopic biopsy spoon has value mainly in biopsy from the upper lobe bronchi. I do not advise using it for biopsy from the esophagus, as the likelihood of injuring the mucous membrane is more than with ordinary biopsy forceps because of the differences in the anatomic features of the bronchus and the esophagus.

Apart from the use of this instrument in biopsy, the handle can accommodate cauterizing ends for high frequency currents (diathermy). This is of value in cases with lesions that need intrabronchial or intraesophageal electrocoagulation; for example, in excessive intrabronchial bleeding, following biopsy, which does not respond to ordinary measures.

In cases of bronchial stenosis due to healed tuberculous ulcerative bronchial lesions, I do lineal cauterizations at right angles to the fibrous tissue bands forming the stenosis. This causes the formation of regular bands of fibrous tissue

Chief ear, nose and throat surgeon-bronchoscopist of the Chest Hospitals, Ministry of Public Health, Cairo, Egypt.

Presented as a *New Instrument* at the Sixtieth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1955, Chicago.

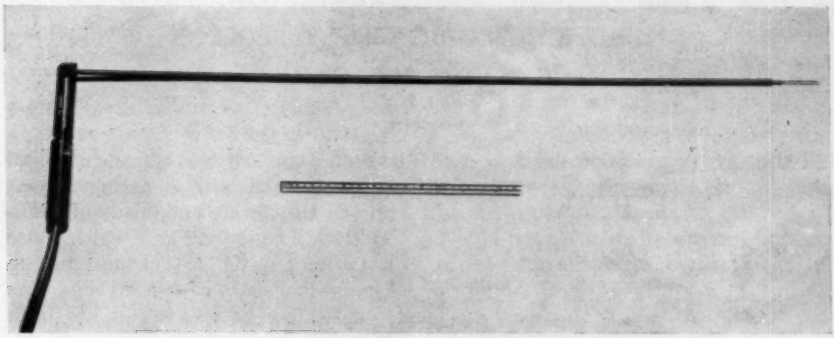

FIG. 1—Bronchoscopic biopsy spoon.

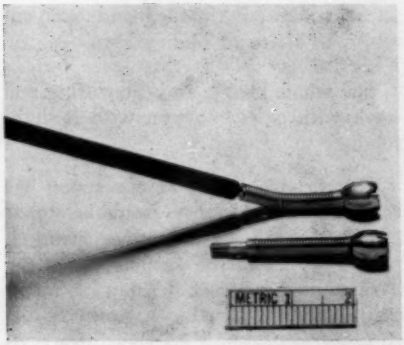

FIG. 2—Cutting end with champagne-cup shape and castellated edge.

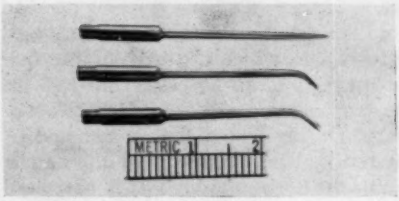

FIG. 3—Cauterizing ends.

which pull on the original bands, resulting in widening the stenosed lumen of the bronchus in moderate degree.

This procedure should be done by expert hands, and for obtaining more successful permanent results, pneumatic bronchial dilatation should be carried out not more than seven days before the date of cauterization. For this purpose I have designed different cauterizing ends, conforming to the direction and shape of the stenosis (fig. 3).

With regard to esophageal lesions, one use of this instrument is in the cauterization of varicose veins in the distal third of the esophagus, which sometimes result in severe hemorrhage.

The lumen of an adult bronchoscope or esophagoscope easily accommodates both the instrument and the suction tube, so that all the manipulations can be done under direct vision, making the procedure very safe and accurate.

Dr. H. J. Moersch, head of the bronchoesophageal section at the Mayo Clinic, was interested in the instrument and had a copy made of it. It is under his clinical trial.

I am grateful to the Mayo Clinic for their assistance in making lantern slides.

TRANSACTIONS

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HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
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All communications regarding the TRANSACTIONS should be addressed to
William L. Benedict, M.D., LL.D., Executive Secretary-Treasurer,
American Academy of Ophthalmology and Otolaryngology,
100 First Avenue Building, Rochester, Minnesota

CLINICS IN MEXICO

At the Clinics in Mexico last October we were privileged to see patients with unusual eye diseases. In one hospital there were demonstrated to us at one time seven patients with the rare disease of the eyes and skin known as the Vogt-Koyanagi syndrome and Harada's disease. Such cases are seldom seen in the United States. We were also shown two patients with Rothmund's syndrome, a rare disease occurring in children in which congenital cataracts are also found. There were many other unusual eye conditions demonstrated to our group of visiting doctors. A very interesting demonstration of fundus photographs was enjoyed by the Academy group in the Hospital for the Prevention of Blindness. A large number of photographs of rare eye conditions were shown and discussed by the hospital staff.

The announcement that Clinics in Mexico City will again be offered following the Chicago meeting has received a very enthusiastic response. Your deposit for the visit automatically registers you for the Clinics. Are you going? Clinic bookings are ahead of those at this time last year.

For those unable to go to Chicago, registration for the Clinics in connection with the land travel will be possible in Mexico and arrangements will be so made.

Ask your questions of Leon V. Arnold, 33 Washington Square West, New York 11. You may have a special problem. He will give you full information promptly. Send your deposit to Mr. Arnold also.

On Tuesday afternoon, on the way to the Pyramids of the Sun and Moon, we shall visit the ruined Monastery of Acolman, founded by the Augustinian friars in 1539. The building is an interesting mixture of medieval and Renaissance architectures. At San Juan Teotihuacan, the Mexican government, in recent years, has begun to rebuild the pyramids and to excavate other buildings and chambers. The towering pyramids will make us appreciate the skill of the people who lived here many centuries ago. The Temple of Quetzalcoatl, the legendary white god, and the vast Citadel will cause us to wonder how this great civilization could have reached such an abrupt and mysterious end.

Later, to contrast with the pagan sights, we shall visit the Basilica of Guadalupe, which is the Mecca of the millions of Catholic faithful in Mexico. We shall see all parts of the great Sanctuary, including the robe of the humble peasant on which appeared the miracle printing of the Virgin.

On our two-days' motor tour, Wednesday and Thursday, we shall drive through the "Desert of the Lions," which is not a desert and has no lions. In the bougainvillaea-draped suburb of San Angel, we shall see the lovely homes of Mexico City's upper class; we shall drive through the very modern residential section of the "Pedregal," built on the lava flow from the long extinct "Xitle"; and we shall visit Mexico's pride in modern architecture, University City, the highlight of which is the library with its outside walls completely covered with mosaics.

Cuernavaca is a favorite resort of residents of Mexico City; here they find a moderate altitude and an even and temperate climate all the year. A stop will be made at the Borda Gardens, where Maximilian and Carlota passed so many idyllic days. We shall see the Cathedral and the Palace of Cortez and have lunch at Hacienda Vista Hermosa, a lovely resort. Driving through the large fields of sugar cane and rice, we shall soon come to the Sierra Madre mountains where Taxco, the town where time stands still, is set like a jewel enfolded by the green-clad slopes.

Romantic Taxco, famous in history and legend, has all its original colonial charm. The cobblestone streets, the overhanging tile roofs, and balconies rising tier upon tier are just as they were centuries ago. The whole city of Taxco is a national monument. Not a building can be repaired, no bit of cobbled pavement can be relaid, nor can a broken roof tile be replaced without the approval of the Commission. The narrow streets are lined with scores of little shops where silver is being fashioned by hand into beautiful jewelry of Aztec and modern designs. The famous church of Santa Prisca, with its twin towers of pink stone, is a highlight.

The night will be spent at Hotel Rancho Telva. It was named for the Metropolitan Opera star, Marion Telva, who has been a frequent visitor. The hotel is an old Spanish-Moorish structure with red-tiled roofs, rambling halls and rough-beamed rooms, to which were added American kitchens and modern bathrooms. There will be time to shop on Thursday morning, and in the afternoon we shall return to Mexico City by the Express Thruway.

See the July-August issue of TRANSACTIONS for details of our Friday tour to Hacienda San Miguel Regla, the Pan-American Doctors Club.

In Memoriam

William Anthony Defnet.....Detroit, Michigan
February 1, 1956

Fayette Clay Ewing.....Pineville, Louisiana
April 15, 1956

Henry Joseph Feaster.....Brooklyn, New York
March 8, 1956

Henry C. Haden.....Houston, Texas
April 25, 1956

Edwin Perry Hall.....Oneota, New York
April 9, 1956

William F. Hughes.....Indianapolis, Indiana
April 1956

Arnold Knapp.....New York, New York
February 29, 1956

Robert Scott Lamb.....Washington, D. C.
March 4, 1956

James Devote Perdue.....Mobile, Alabama
May 18, 1956

Samuel B. Westlake.....St. Louis, Missouri
March 27, 1956

FINANCIAL REPORT

Annual Audit for Calendar Year — 1955

May 16, 1956

American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota
Gentlemen:

We have examined the balance sheet of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, Rochester, Minnesota, as of December 31, 1955, and the related statements of operations and net worth for the year then ended. Our examination was made in accordance with generally accepted auditing standards and included such tests of the accounting records and such other auditing procedures as we deemed necessary in the circumstances. We submit herewith our report, which includes the following statements:

Exhibit

"A"—Balance Sheet, December 31, 1955
"B"—Statement of Operations, Year ended December 31, 1955
"C"—Analysis of Funds from Special Contributions
"D"—Analysis of Net Worth Account
"E"—Analysis of Increase in Market Value of Securities

Schedule

1—Securities owned, December 31, 1955

FINANCIAL CONDITION

A balance sheet as at December 31, 1955, appears in Exhibit "A." The nature of the various assets and liabilities appearing on this balance sheet is described in the following comments.

Current Assets, $72,607.07

Cash on deposit at the various banks, totaling $47,503.24, was verified by direct correspondence with the depositories. The funds in the Canadian bank are stated in U. S. dollars. Petty cash fund of $25.00 was verified by actual count during the course of our examination.

Membership dues receivable, in the amount of $1,720.00, consists of delinquent dues for 1955 and prior years which were verified from the records only. Other receivables, taken from the records without further verification, consist of amounts due from members and others in the sum of $708.75 for slides, abstracts, manuals and monographs, and $598.02 due from various firms for advertising placed in the TRANSACTIONS.

The inventory of abstracts, manuals and monographs, Bound Volumes, slides and film strips, in the amount of $22,033.91, was taken from the inventory records as prepared by employees of the Academy without further verification except for testing of prices, extensions and footings. The major portion of this inventory is held by the Academy's printer at Omaha, Nebraska, shipments being made as directed by the Academy.

Investments, $223,843.60

In Schedule No. 1, we present a schedule of bonds and stocks which had a market value of $223,843.60 on December 31, 1955. U. S. Savings Series "G" Bonds are stated at maturity values rather than at current redemption prices.

These securities are held for safekeeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, where they were inspected by us. Interest earned on bonds since the dates on which last payments became due has not been accrued. As shown in Exhibit "E," the market value of these securities increased $13,040.98 during the year.

Fixed Assets, $116,353.60

Furniture, fixtures and equipment amounting to $12,735.68 represents equipment acquired by the Academy over a period of years. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried and appraised at a market value of $1,420.28 as of January 1, 1944, and set up on the books for that amount. Subsequent purchases of equipment have been verified through an examination of supporting vouchers, with the additions during the current year amounting to $959.79. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Expenditures for motion picture films and slides, entitled "Embryology of the Eye," totaling $40,900.00, and "Embryology of the Ear," with a production cost of $101,462.17, represents the cost to complete these medical motion picture films and slides by Sturgis-Grant Productions, Inc. Prints made from the

master film are available for sale at approximate cost. The cost of the original films and slides is being amortized at a rate that appears to be adequate.

Deferred Charges, $20,957.27

Unexpired insurance premiums as computed by us amounted to $302.82.

Prepaid postage deposit of $151.20, verified by correspondence, represents the balance of cash advanced to the U. S. Post Office at Omaha, Nebraska, to be used as postage on subsequent mailings of the TRANSACTIONS and supplements.

Supplies in the amount of $503.25, as inventoried by employees without further verification by us, consists of lapel buttons which are awarded to Senior Fellows.

Payments totaling $20,000.00 have been made to the National Academy of Sciences, acting as fiscal agent for the American Registries of Pathology. The total cost of the project will be approximately $46,600.00. The Academy will be reimbursed in part or in full, when funds become available from the sales of this atlas for which the American Registries of Pathology will be the distributor.

Current Liabilities, $40,351.95

Accrued expenses of $35,462.57 represents unpaid December 1955 printing costs of the TRANSACTIONS, cost of manuals and plaques, and other office expenses, totaling $6,453.65; expenses incurred in connection with the annual meeting of the Academy held at Chicago in October 1955, amounting to $28,132.83, and other office expenses totaling $876.09. Accrued social security and withholding taxes unpaid as of December 31, 1955, amounted to $596.38. The Sub-Committee on Noise in Industry did not withhold social security taxes from wages paid to their employees during 1955; furthermore the Committee did not pay any social security or state unemployment taxes in 1955 on these salaries, and these liabilities are not shown on the balance sheet.

Prepayments for Bound Volumes, etc., totaling $4,098.00, consists of deposits made by members and others for Bound Volumes and various periodicals to be delivered in 1956.

Deferred income, membership dues, totaling $195.00, represents dues paid in advance by Academy members.

Undisbursed Funds from Special Contributions, $5,521.16

Undisbursed funds from contributions received and designated for "Specific Projects" are analyzed in Exhibit "C," which shows 1955 transactions as well as prior year balances.

Net Worth, $387,888.43

As shown in Exhibit "D," the net worth of the Academy increased from $320,595.21 on December 31, 1954, to $387,888.43 on December 31, 1955. Of this increase, $54,252.24 represents the net income for the year, as shown in Exhibit "B," and $13,040.98 consists of the increase in market value of securities, as explained in Exhibit "E."

OPERATIONS

A statement of operations for the year ended December 31, 1955, is presented in Exhibit "B." A summary of this statement and a comparison with the results for the year 1954 is as follows: [See accompanying table, page 504.]

GENERAL

Upon examination of the insurance policies in force, we found fire and extended coverage on furniture, fixtures and equipment in the amount of $7,500.00 with 80% co-insurance. Paper stock, publications, etc., in custody of the printer at Omaha, Nebraska, are insured for $12,000.00 with 80% co-insurance. This coverage has since been increased to $18,000.00 to meet 80% co-insurance requirements. Comprehensive general liability insurance is in force, covering bodily injury in the amount of $25,000.00 for each person and $25,000.00 for each accident. This coverage increases to $50,000.00 for each person and $100,000.00 for each accident during the coming annual convention at the Palmer House, Chicago, Illinois.

Property damage liability is covered in the amount of $5,000.00 for each accident with aggregate total coverages of $25,000.00.

Workmen's compensation insurance, which is being carried at the statutory amounts, includes coverage for all Academy employees, including additional help hired at the annual convention.

The Executive Secretary-Treasurer of the Academy is bonded for $25,000.00 and the manuscript editor for $2,500.00. Other principal office employees are bonded for $1,000.00 each. It is our opinion that the office employees fidelity bond coverage of $1,000.00 is inadequate in view of the large amounts of cash handled at convention time.

The loss of money, securities and other property is covered up to $2,400.00 within the premises for safe burglary, and $2,100.00 outside the premises against messenger robbery.

	Year 1955	*Year* 1954	*Increase* **Decrease**
Revenues	$257,538.66	$250,533.62	$ 7,005.04
Operating Expenses:			
General Administrative	65,396.77	79,645.63	**14,248.86**
TRANSACTIONS	60,887.25	68,237.03	**7,349.78**
Home Study Courses	7,691.49	8,695.80	**1,004.31**
Convention expense	52,471.57	74,950.75	**22,479.18**
Educational Fund	11,894.74	4,445.19	7,449.55
Total operating expenses	198,341.82	235,974.40	**37,632.58**
Operating Income	59,196.84	14,559.22	44,637.62
Other Income:			
Gain on sale of securities, etc.	35.00	781.75	**746.75**
Total	59,231.84	15,340.97	43,890.87
Other Deductions:			
Loss, worthless securities	4,979.60		4,979.60
Net Income	$ 54,252.24	$ 15,340.97	$ 38,911.27

At the annual convention the coverage for loss within the premises on money, securities and other property is increased to $40,000.00, with a limit of $20,000.00 on money.

We acknowledge the very courteous assistance extended us by Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, and his staff during the progress of this examination.

OPINION

In our opinion, subject to the comments in the preceding paragraphs, the accompanying balance sheet and related statements of operations and net worth present fairly the financial position of the American Academy of Ophthalmology and Otolaryngology at December 31, 1955, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,
BYERS, WOBSCHALL & MILLER
Certified Public Accountants
By Edw. A. Wobschall, C.P.A.

EXHIBIT "A"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Balance Sheet as at December 31, 1955

ASSETS

Current Assets:			
Cash, First National Bank, Rochester, Minnesota		$29,324.12	
Cash, First National Bank, Minneapolis, Minnesota		17,095.24	
Cash, Bank of Montreal, Toronto, Canada		1,083.88	
Petty cash fund		25.00	
Membership dues receivable, delinquent		1,720.00	
Accounts receivable, others		1,306.77	
Checks held for collection		18.15	
Inventory, abstracts, manuals, monographs, films, etc.		22,033.91	
Total current assets			$ 72,607.07
Investments: (Schedule No. 1)			
Bonds and stocks			223,843.60
Fixed Assets:			
Furniture and fixtures	$ 12,735.68		
Less reserve for depreciation	7,014.75	5,720.93	
Embryology of the Eye: Film & Slides	40,900.00		
Less reserve for depreciation	24,120.00	16,780.00	
Embryology of the Ear: Film & Slides	101,462.17		
Less reserve for depreciation	7,609.50	93,852.67	116,353.60
Deferred Charges:			
Prepaid insurance		302.82	
Prepaid postage		151.20	
Supplies		503.25	
Atlas of Otolaryngic Pathology		20,000.00	20,957.27
Total Assets			$433,761.54

LIABILITIES AND NET WORTH

Current Liabilities:		
Accrued expenses	$35,462.57	
Accrued payroll taxes	596.38	
Prepayments for Bound Volumes, etc.	4,098.00	
Deferred income, membership dues	195.00	
Total current liabilities		$ 40,351.95
Undisbursed Funds from Special Contributions: (Exhibit "C")		
Wherry Memorial Fund		5,521.16
Net Worth (Exhibit "D")		387,888.43
Total Liabilities and Net Worth *		$433,761.54

* Subject to report comments

EXHIBIT "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Operations
Year Ended December 31, 1955

Revenues:			
Membership dues			$ 66,753.00
Membership applications			6,192.00
Abstracts, manuals and monographs			9,214.04
Certificates			1,532.50
Films and slides			1,913.50
Testing and calibration of tonometers			1,157.00
Royalties, Atlas and Textbook of Ophthalmic Pathology			833.40
Convention			86,621.00
Home Study Courses			7,204.00
TRANSACTIONS			66,164.40
Income from securities:			
Interest, Educational Fund		$ 7,233.75	
Dividends, Educational Fund		2,702.59	9,936.34
Other income			17.48
Total Revenues			257,538.66
Operating Expenses:			
General administrative		65,396.77	
TRANSACTIONS		60,887.25	
Home Study Courses		7,691.49	
Convention expenses		52,471.57	
Educational Fund:			
Trustee fees	$ 150.24		
Depreciation, films and slides	11,744.50	11,894.74	
Total Operating Expenses			198,341.82
Operating Income			59,196.84
Other Income:			
Gain on sale of equipment			35.00
			59,231.84
Other Deductions:			
Loss on worthless securities			4,979.60
Net Income			$ 54,252.24

EXHIBIT "C"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Analysis of Funds from Special Contributions

WHERRY MEMORIAL FUND		
Balance, December 31, 1954		$ 5,407.72
Receipts, Year 1955:		
Interest on U. S. Savings Bonds	$ 65.00	
Dividends	48.44	113.44
Balance, December 31, 1955		$ 5,521.16
SUB-COMMITTEE ON NOISE IN INDUSTRY FUND		
Balance, December 31, 1954		None
Receipts, Year 1955:		
Contributions received from outside sources		$ 76,835.24
Total		76,835.24
Less:		
Transfers to Sub-Committee on Noise in Industry, Los Angeles, California *		76,835.24
Balance, December 31, 1955		None

* This amount was received by the Sub-Committee on Noise in Industry as shown in audit report (not certified) submitted to the Academy. This Sub-Committee on Noise in Industry has an unexpended balance of $12,321.11, which is not shown on balance sheet marked Exhibit "A" in this report.

EXHIBIT "D"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Analysis of Net Worth Account
For Year Ended December 31, 1955

Net Worth, December 31, 1954		$320,595.21
Additions, Year 1955:		
Net income, year ended December 31, 1955 (*Exhibit "B"*)	$54,252.24	
Increase in market value of securities during 1955 (Exhibit "E")	13,040.98	67,293.22
Net Worth, December 31, 1955		$387,888.43

EXHIBIT "E"

Analysis of Increase in Market Value of Securities

	Cost	*Market Value*	*Increase* **Decrease**
December 31, 1954	$189,707.21	$213,042.49	$ 23,335.28
December 31, 1955	187,467.34	223,843.60	36,376.26
Difference	**$2,239.87**	$ 10,801.11	$ 13,040.98

SCHEDULE No. 1

American Academy of Ophthalmology and Otolaryngology
Securities Owned, December 31, 1955

EDUCATIONAL FUND

BONDS	RATE %	MATURITY	PAR VALUE	COST	MARKET VALUE*
Citizens Building Co. of Cleveland, 1st Leasehold Sinking Fund	6	1/ 1/46	$ 5,000.00	None (1)	None
Van Swerigen Co. Certificates of Indebtedness, Series 1925	6	12/31/48	6,000.00	$ 1,617.03 (2)	$ 980.00
Dominion of Canada, 8th Victory Loan Series P5	3	10/ 1/63	7,500.00	7,612.50 (3)	7,265.25
Dominion of Canada, 9th Victory Loan Series P7	3	9/ 1/66	2,000.00	1,880.00 (3)	1,925.00
Government of Canada	3¼	10/ 1/79	3,500.00	3,629.84 (3)	3,386.25
U.S.A. Savings, Series G	2½	3/ 1/56	6,000.00	6,000.00	6,000.00
U.S.A. Savings, Series G	2½	5/ 1/56	2,000.00	2,000.00	2,000.00
U.S.A. Savings, Series G	2½	11/ 1/56	25,000.00	25,000.00	25,000.00
U.S.A. Savings, Series G	2½	7/ 1/58	30,000.00	30,000.00	30,000.00
U.S.A. Savings, Series G	2½	12/ 1/58	25,000.00	25,000.00	25,000.00
U.S.A. Savings, Series G	2½	3/ 1/59	15,000.00	15,000.00	15,000.00
U.S.A. Savings, Series G	2½	7/ 1/60	30,000.00	30,000.00	30,000.00
Total Bonds				$147,739.37	$146,556.50

STOCKS	KIND	PAR VALUE	SHARES		
Citizens Building Co. of Cleveland	Shares	No Par	50	None	None
Commonwealth Edison Co.	Common	$25.00	100	$ 2,294.56	$ 4,125.00
E. I. DuPont De Nemours & Co.	Common	5.00	60	2,347.31	13,866.00
Louisville Gas & Electric Co.	5% Preferred	25.00	200	5,450.00	6,100.00
Massachusetts Investors Trust	Trust Cert.	1.00	1,264	18,609.66	42,217.60
Ophthalmic Publishing Co.	Capital	20.00	5	None	100.00
Northern Illinois Gas Co.	Common	5.00	34	401.44	654.50
Central Maine Power Co.	Common	10.00	200	5,225.00	4,824.00
Total Stocks				$ 34,327.97	$ 71,887.10
TOTAL EDUCATIONAL FUND				$182,067.34	$218,443.60

WHERRY MEMORIAL FUND

	KIND	PAR VALUE	SHARES		
Rochester Building & Loan Assn.	Capital	$100.00	54	5,400.00	5,400.00
TOTAL INVESTMENTS				$187,467.34	$223,843.60

* Except for U.S.A. Savings, Series G Bonds, which are included at maturity values
(1) Charged off in 1955. No market available
(2) In process of liquidation
(3) In U. S. dollars

SECTION ON INSTRUCTION

SIXTY-FIRST ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 14-19, 1956

PALMER HOUSE

Chicago, Illinois

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1956 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 12.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Orders will not be accepted for presale after October 4. To ensure acceptance they should be mailed by October 1 to William L. Benedict, M.D., 100 First Avenue Building, Rochester, Minnesota.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two different days, allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course runs two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third choices for each period in case first choice is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

Tickets will be on sale during the meeting at the Instruction Desk in the Registration Foyer.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Adler	Physiology of Vision	3 PERIODS M-4, T-4, W-4
2	Allen et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
3	Ballintine & Johnson	Clinical Aqueous Tonography	2 PERIODS M-5, M-6
4	Becker & Kinsey	Studies on Aqueous Flow	2 PERIODS M-4, M-5
5	Bedell	Medical Ophthalmoscopy	3 PERIODS M-4, T-4, W-4
6	Berke	Surgery of Oblique Muscles	2 PERIODS M-4, M-5
7	Berliner	Slit Lamp Microscopy: Advanced	4 PERIODS M-4, T-4, W-4, Th-4
8	Boeder	Practical Optics	2 PERIODS T-4, T-5
9	Brockhurst	Indirect Ophthalmoscopy	2 PERIODS T-5, T-6
10	Brown et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
11	Byrnes	Ophthalmic Filters	2 PERIODS T-4, T-5
12	Clark, G.	Retinal Detachment	3 PERIODS M-6, T-6, W-6
13	Clark, W.	Cataract Extraction: Selection of Patient	2 PERIODS M-5, M-6
14	Clark, W.	Surgery of Ocular Muscles	2 PERIODS T-5, T-6
15 or 16	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
17	Costenbader et al	Comitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
18	Guibor	Orthoptics: Office and Home Techniques	3 PERIODS T-6, W-6, Th-6
19	Henderson	Neuro-ophthalmology	3 PERIODS M-5, T-5, W-5
20	Hollenhorst	Arterial Diseases	2 PERIODS W-5, W-6
21	Hughes et al	Ophthalmic Plastic Surgery: Fundamental	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
22	Knighton et al	Highlights in Glaucoma	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
23	McLean et al	Retinal Detachment	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
24	Ogle	Essential Aspects of Binocular Vision	2 PERIODS T-4, T-5
25	O'Rourke	Cross Cylinders & Astigmatic Dials	2 PERIODS M-4, M-5
26	Pfeiffer & Jones, I.	Roentgenography of the Eye & Orbit	2 PERIODS M-4, M-5

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
27	Pischel	Retinal Detachment	3 PERIODS M-6, T-6, W-6
28	Regan, J., & Regan, C.	Refraction: Practical Aspects & Aids in Difficult Problems	6 PERIODS M-4, M-5, T-4, T-5 W-4, W-5
29	Rolett	Gonioscopy: Practical Application	2 PERIODS W-4, W-5
30	Romaine et al	Ocular Motility	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
31	Romaine	Refraction Problems: Treatment	3 PERIODS M-6, T-6, W-6
32	Sanders et al	Vascular Diseases	3 PERIODS T-6, W-4, Th-4
33	Schimek & Bousquet	Gonioscopy & Aqueous Outflow Determinations	2 PERIODS M-5, M-6
34	Sloane	School Vision Testing	2 PERIODS M-4, M-5
35	Sloane	Telescopic Lenses	2 PERIODS T-4, T-5
36	Pollen & Gettes	Prescription of Prisms; Drugs in Refraction	2 PERIODS W-4, W-5
37	Smith et al	Ophthalmic Plastic Surgery: Advanced	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
38	Stow	Diagnostic & Therapeutic Office Procedures	2 PERIODS W-4, W-5
39	Tait	Heterophoria	2 PERIODS M-4, M-5
40	Theobald et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
41	Thorpe et al	Slit Lamp Biomicroscopy: Fundamentals	9 PERIODS M-4, M-5, M-6, T-4, T-5 T-6, W-4, W-5, W-6
42	Thorpe et al	Slit Lamp Biomicroscopy: Recent Developments	3 PERIODS Th-4, Th-5, Th-6
43	Thorpe	Surgery of Intraocular Foreign Bodies	2 PERIODS T-4, T-5
44	Townes	Cataract Surgery: Complications	2 PERIODS W-4, W-5
45	Troutman	Enucleation Surgery: Management	2 PERIODS T-4, T-5
46	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-4, M-5, T-4, T-5 W-4, W-5
47	Wise	Retinal Neovascularization	2 PERIODS W-4, W-5
48	Home Study		10 PERIODS M-4, M-5, M-6, T-4, T-5 T-6, W-4, W-5, W-6, Th-4

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Orders will not be accepted for presale after October 4. To ensure acceptance they should be mailed before October 1 to

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

Academy Candidates and Guests May Purchase Tickets Only at Instruction Desk in Registration Foyer

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 12

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.

2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1956 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.

3. Make check payable to W. L. Benedict, M.D.

4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD		FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
		Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON.	4						
P.M.	5						
	6						
TUES.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
WED.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
THURS.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Emotional Problems in the Ophthalmologist's Patients
204	Alvaro	Uveitis: Etiology & Treatment
207	Baird	Lesions of the Orbit: Differential Diagnosis
208	Bannon	Aniseikonia: Its Role in Refractive Problems
213	Berens	New Procedures in Ophthalmic Surgery
215	Bettman	Value of Goniotomy
217	Boyes	Problems in Eye Surgery
223	Chamlin	Perimetry
226	Cooper	Principles of Muscle Surgery
240	Dunlap	Divergence Anomalies
242	Falls	Practical Application of Heredity
244	Fink	Binocular Problems in Routine Refraction
245	Fonda	Subnormal Vision: Technique for Examination
267	Jacobson	Electroretinography
273	Katz	Ophthalmic Anatomy
291	Linn	Paralyses of Motor Nerves to the Eye
313	Myers	Vertical Phorias & Tropias
350	Whalman	Practical Points in Eye Surgery

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
203	Allen, H.	Operating Room Technique
210	Benedict	Atrophic Changes of the Iris
216	Block	Office Practice of Ophthalmology
218	Breinin	Electromyography
229	Coston	Infiammatory & Degenerative Disease of the Retina
235	Danielson	Retinoblastoma
248	Fry	Lesions at Posterior Pole of Eye
256	Hague	Irradiation in Ophthalmology
258	Hanno	Internal Medicine: Ophthalmic Problems
259	Hanser	Studies in Ocular Allergy
266	Iliff	Ptosis
276	Keyes	Retinal Vascular Disease
307	Merriam	Radiotherapy
348	Walsh	Neurological Syndromes

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
205	Amenabar	Retinal Detachment: Localization & Closure of Tears
206	Atkinson	Selection of Procedure for Cataract Extraction
211	Benton	Refraction: Handling the Difficult Patient
219	Calhoun	Dystrophies & Degenerations of the Cornea
225	Cohoe	Special Reading Instruction for the Partially Seeing
227	Cordes	Eye Changes in Diabetes
233	Cutler	Cataract: Methods of Extraction
234	Daily	Biomicroscopy of Vitreous & Fundus Oculi
236	Davidson	Surgical Instruments: Preparation & Care
237	Davis	Sutures in Cataract Surgery
238	Diskan	Visual Screening in Schools
246	Fralick	Surgical Anatomy in Operations for Glaucoma
247	Fread	Ideal Refraction Equipment
255	Gump	Recurrent Iridocyclitis
269	Jardon	Fitting Ocular Prostheses
272	Kasper	Dacryocystorhinostomy
274	Kennedy	Practical Perimetry
277	Kirby	Problems in Presbyopia
283	Leinfelder	Papilledema & Optic Neuritis
297	MacLean	Surgical Management of Cataracts
299	Mangold	Proper Fitting of Glasses
312	Murrah	Aids for Subnormal Vision
318	Perera	Retinal Detachment
325	Reese	Orbital Tumors
345	Uihlein	Neuralgias of the Head

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
204	Alvaro	Uveitis: Etiology & Treatment
207	Baird	Lesions of the Orbit: Differential Diagnosis
217	Boyes	Problems in Eye Surgery
228	Cordes	.....SOLD OUT.....
239	Dixon	Indirect Ophthalmoscopy
240	Dunlap	Divergence Anomalies
241	Egan	Cross Cylinder: Application & Theory
243	Falls	Ocular Geriatrics
244	Fink	Binocular Problems in Routine Refraction
249	Gaines	Ophthalmologic Examination from a Neuro-ophthalmologic Viewpoint
270	Jones, L.	The Lacrimal Drainage System
289	Lewis	External Diseases
294	Lowrey	Tropical Ophthalmology
298	Macnie	Head Trauma with Ocular Symptoms
303	Maumenee	Surgical Indications for Corneal Procedures
310	Morrison	Office Management of Glaucoma
316	Paulson	Lighting for Comfortable & Accurate Seeing

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
218	Breinin	Electromyography
250	Gartner	Fundus Diseases
251	Gifford	Surgical Procedures on Lacrimal Apparatus
256	Hague	Irradiation in Ophthalmology
260	Harley	Lesions of 3rd, 4th and 6th Cranial Nerves
263	Hester	Clinical Photography
268	Jampolsky	Ocular Divergence
271	Joy	Cerebral Angiography
282	Leahey	Corneal Transplantation
290	Linksz & Tait	Telescopic Lenses & Magnifying Systems
309	Miller	Corneal Disease
319	Pino & Edwards	Correction of Aphakia
326	Rizzuti	Tumors of the Lids & Ocular Adnexa
330	Rychener	The Lacrimal Apparatus
342	Swan	Advances in Ophthalmic Therapy

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
212	Berens	Cyclodiathermy, Cycloelectrolysis, Iridencleisis & Cyclodialysis
219	Calhoun	Dystrophies & Degenerations of the Cornea
224	Chandler	Cataract Surgery: Complications
231	Cunningham	Types of Head Pain
233	Cutler	Cataract: Methods of Extraction
234	Daily	Biomicroscopy of Vitreous & Fundus Oculi
247	Fread	Ideal Refraction Equipment
252	Gipner	Recent Advances in Ophthalmology
274	Kennedy	Practical Perimetry
277	Kirby	Problems in Presbyopia
279	Kornzweig	Practical Embryology
284	Leinfelder	Perimetry & Use of Tangent Screen
292	Lloyd	Diseases of Cornea
295	Lubkin	Psychosomatic Aspects of Ophthalmology
296	Luce	Wound Healing
299	Mangold	Proper Fitting of Glasses
304	McDonald	Value of Tinted Lenses
318	Perera	Retinal Detachment
327	Roberts	Early Diagnosis of Glaucoma
329	Ruedemann	Physiologic Changes Due to Vertical Muscles
331	Scarney	Slit Lamp Biomicroscopy
332	Schaie	Surgery for Congenital Glaucoma
345	Uihlein	Neuralgias of the Head

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
220	Cassady	Dacryocystitis & Lacrimal Drainage
222	Chamberlain	Vertical Motor Anomalies
232	Cusick	Ptosis
239	Dixon	Indirect Ophthalmoscopy
241	Egan	Cross Cylinder: Application & Theory
257	Haik	Intracapsular Cataract Extraction
262	Havener	Abnormalities of the Optic Disc
275	Kestenbaum	Chiasmal Diseases
278	Klien	Clinical Pathology of the Retina
281	Kronfeld	Choice of the Surgical Procedure in Glaucoma
285	Lemoine	Vitreous Opacities
288	Levy	Motion Picture Photography
289	Lewis	External Diseases
306	McGuire	Heterophoria & Heterotropia
310	Morrison	Office Management of Glaucoma
311	Mullen	Ophthalmic Emergencies
340	Steffensen	ACTH, Cortisone & Hydrocortisone
343	Tassman	Retinopathy in Diabetes

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
209	Haas	Congenital Glaucoma: Diagnosis & Treatment
251	Gifford	Surgical Procedures on Lacrimal Apparatus
253	Givner	Eye Changes in Systemic Disease
261	Harris	Anesthetic Agents & Techniques
265	Hughes	Advances in Diagnosis & Surgical Treatment
280	Kronenberg	Practical Ocular Therapeutics
282	Leahey	Corneal Transplantation
290	Linksz & Tait	Telescopic Lenses & Magnifying Systems
293	Lloyd	Fundus Lesions of the Senile Eye
300	Mann, M.	Hypertensive Vascular Changes in the Fundus
302	Marshall	Chronic Infections of the Lids
315	Olson	Iritis, Iridocyclitis & Keratitis
323	Prewitt	External Eye Diseases Associated with Allergy
330	Rychener	The Lacrimal Apparatus
337	Solanes	Uveomeningitis Syndromes
344	Theodore	Ocular Therapeutics
352	Wood	Inflammatory Central Retinal Lesions

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
205	Amenabar	Retinal Detachment: Localization & Closure of Tears
221	Cassady	Toxoplasmic Retinochoroiditis
252	Gipner	Recent Advances in Ophthalmology
254	Gordon	Visual Aids
264	Hogan	Lesions of the Choroid
284	Leinfelder	Perimetry & Use of Tangent Screen
287	Lepard	Eye Diseases Common to Children
301	Mann, W.	Functional Visual Impairment
305	McGraw	Contact Lenses
308	Meyer	Surgical Treatment of Glaucoma
314	Nethercut	Chorioretinitis: Etiology & Treatment
317	Payne	Extraction of Complicated Cataract
320	Pino & Edwards	Problems in Bifocals
322	Post	Refinements of Refraction
324	Randolph	Advanced Glaucoma
328	Rudolph & Schlaegel	Chemotherapy
332	Scheie	Surgery for Congenital Glaucoma
336	Simpson	Probing of the Nasolacrimal Duct
339	Spaeth	Plastic Surgery: Practical Aspects
341	Sugar	Office Diagnosis of Glaucoma
346	Vail	Surgery of the Senile Cataract
347	Veirs	External Dacryocystorhinostomy
349	Weisser	Chronic Uveitis
351	Wise	Fundus Change of the Optic Nerve Head

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alfano	Internal Carotid Artery
220	Cassady	Dacryocystitis & Lacrimal Drainage
222	Chamberlain	Vertical Motor Anomalies
230	Cowen	Office Technique for Simplified Gonioscopy
232	Cusick	Ptosis
259	Hanser	Studies in Ocular Allergy
275	Kestenbaum	Chiasmal Diseases
278	Klien	Clinical Pathology of the Retina
285	Lemoine	Vitreous Opacities
287	Lepard	Eye Diseases Common to Children
298	Macnie	Head Trauma with Ocular Symptoms
306	McGuire	Heterophoria & Heterotropia
311	Mullen	Ophthalmic Emergencies
321	Posner	Glaucomatocyclitic Crises
331	Scarney	Slit Lamp Biomicroscopy
333	Schwartz	Eye Changes Associated with Skeletal Disease
335	Simpson	Convergence Insufficiency & Divergence Excess
338	Spaeth	Selection of Surgery for Complicated Ptosis
340	Steffensen	ACTH, Cortisone & Hydrocortisone
343	Tassman	Retinopathy in Diabetes

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
209	Haas	Congenital Glaucoma: Diagnosis & Treatment
214	Bettman	Radioactive Isotopes
260	Harley	Lesions of 3rd, 4th & 6th Cranial Nerves
280	Kronenberg	Practical Ocular Therapeutics
286	Lennon	Therapy of Acute Inflammatory Glaucoma
300	Mann, M.	Hypertensive Vascular Changes in the Fundus
302	Marshall	Chronic Infections of the Lids
308	Meyer	Surgical Treatment of Glaucoma
315	Olson	Iritis, Iridocyclitis & Keratitis
323	Prewitt	External Eye Diseases Associated with Allergy
334	Shipman	Scleral Resection
344	Theodore	Ocular Therapeutics
349	Weisser	Chronic Uveitis
352	Wood	Inflammatory Central Retinal Lesions

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
317	Payne	Extraction of Complicated Cataract
347	Veirs	External Dacryocystorhinostomy

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
101	Brewer & Briess	Voice Therapy	2 PERIODS Th-2, Th-3
102 or 103	Conley, Daly & Martin	Carcinoma of Head & Neck	2 PERIODS T-1, T-2 or W-1, W-2
104	Converse	Nasal Plastic Surgery—Basic	2 PERIODS T-1, T-2
105	Converse	Nasal Plastic Surgery—Advanced	2 PERIODS Th-2, Th-3
106 or 107	Craft	Nasal Allergy	2 PERIODS T-2, T-3 or Th-2, Th-3
108 or 109	Dixon, Sooy & Goodale	Rhinologic Problems	2 PERIODS T-2, T-3 or W-2, W-3
110	Fox et al	Noise in Industry	2 PERIODS Th-2, Th-3
111 or 112	Hansel	Allergy	2 PERIODS W-2, W-3 or Th-1, Th-2
113 or 114	Hardy & Pauls	Clinical Audiometry for Young Children	2 PERIODS T-1, T-2 or W-1, W-2
115	Hilding	Physiology of Hearing: Relation to Hearing Loss	2 PERIODS T-2, T-3
116 or 117	Jackson	Cancer of Larynx, Bronchi & Esophagus	2 PERIODS W-2, W-3 or Th-2, Th-3
118	Kazanjian et al	Traumatic Injuries of Face	2 PERIODS W-2, W-3
119	Lindsay	Vertigo	2 PERIODS Th-1, Th-2
120	Maxwell	Diseases of Salivary Glands	2 PERIODS W-1, W-2
121 or 122	Meltzer et al	Your Otological Office Problems	2 PERIODS T-1, T-2 or W-2, W-3
123 or 124	Mueller et al	What the Otologist Should Know About Hearing Aids	2 PERIODS T-1, T-2 or Th-2, Th-3
125	Reed et al	Voice Therapy	2 PERIODS T-1, T-2
126	Scheldrup	Anatomy of Neck	2 PERIODS Th-2, Th-3
127 or 128	Seltzer	A Typical Rhinoplasty	2 PERIODS T-2, T-3 or Th-2, Th-3
129 or 130	Walsh	What Can Be Done for Deafness	2 PERIODS T-1, T-2 or Th-1, Th-2
131 or 132	Work	Techniques in Wide Field Laryngectomy	2 PERIODS T-2, T-3 or W-2, W-3
133	Harbert	Clinical Audiology	2 PERIODS Th-2, Th-3

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Adin	Approaches to the Mastoid Antrum
402	Albers	Branchial Anomalies
403	Alexander	The Adenoid Problem
406	Anderson	Streamlined Allergy History
410	Baker	Chronic Nonspecific Laryngitis
413	Bell	Harelip Nose in the Adult
419	Cody	Eustachian Tube
425	Davies	Pharynx Development in Man
447	Hantman	Newer Aids in Teaching Otology
449	Harris	Nasal Polypi
453	Holinger & Johnston	Bronchoscopic Techniques
458	Huffman	Treatment of Facial Fractures
465	Keim	Management of Parotid Tumors
475	Lewis, J.	Facial Scars
491	Putney	Combined One-Stage Pharyngeal Diverticulectomy
498	Sellers	Frontal Sinusitis: Acute & Chronic Infections
499	Senturia	Diseases of the External Ear
505	Skolnik	Cleft Palate
512	Tato	Tympanoplasty: Functional Examination
516	Tschiassny	Labyrinthine Function

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
405	Altmann	Tympanoplasty
417	Brodnitz	The Actor & Singer in ENT Practice
424	Crispell	Allergy in Children
436	Forman	Binaural Hearing
450	Higbee	Office Practice
466	Kelemen	Stapes Mobilization
468	Kemler	Bilateral Thyrotomy for Cancer of Larynx
472	Lawrence	Acoustic Tolerance of the Individual Ear
476	Lewis, M.	Temporal Bone Surgery
486	Neivert	Workshop for Deformed Instruments
494	Rinkel	Food Allergy
502	Simmons	Problem of Bleeding Patient
507	Smith	Facial Nerve Paralyses
515	Tremble	Practical Therapeutics in Office Practice
517	Van Alyea	Management of Sinusitis

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
407	Anson & Bast	Anatomy of Ear & Temporal Bone
412	Becker	Plastic Surgery of Protruding Ears
414	Bellucci	Submucous Resection of Nasal Septum
416	Brandenburg	Understanding the Problem Patient
418	Canfield	Speech Audiometry
423	Crane	Helpful Hints
427	Dean	Practical Neuro-otolaryngology
428	Derlacki	Conductive Deafness
434	Ferguson	Pediatric Bronchography
437	Fowler	Facial Paralysis & Tics
456	House	Surgery of Chronically Discharging Ear
461	Jones	Allergic Rhinitis
474	Lempert	Temporal Bone Surgery
482	Miller, J.	Mucolytic Aerosols for Obstructive Respiratory Conditions
485	Morrison	The Pain in the Neck
493	Reger	Practical Office Audiometry
496	Ryan	Headache
508	Som	Laryngoesophagectomy
511	Syracuse	Prevention of Postoperative Drooping of Nasal Tip
520	Wanamaker	Temporomandibular Joint
522	Williams	Treatment of Meniere's Disease
524	Wynn	External Nasal Growths

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Albers	Branchial Anomalies
403	Alexander	The Adenoid Problem
404	Alfaro	Office Design & Equipment
408	Anthony	Atresias of Ear Canal
409	Ashley	Respiratory Allergy
413	Bell	Harelip Nose in the Adult
426	Davison	Ethmoid Sinus Disease
429	DeWeese	Problem of the Dizzy Patient
449	Harris	Nasal Polypi
451	Hilger	The Injured Malar Bone
452	Hilsinger	Vascular Headaches
457	House	Stapes Mobilization Procedure
459	Huffman	Acute Injuries of Soft Tissues of Face
462	Jordan	Nasal Fractures
467	Kelemen	Congenital Deafness
475	Lewis, J.	Facial Scars
480	McNally	Labyrinthine Tests
492	Reed, McLaurin & Senturia	Pathology & Bacteriology
493	Reger	Practical Office Audiometry
501	Shambaugh	Practical Office Routine
506	Skolnik	Electrodiagnosis of Facial Nerve
513	Tato	Tympanoplasty

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
405	Altmann	Tympanoplasty
411	Barrett	Neuroautonomic Aspects of Headache Therapy
420	Corgill	Laryngopharyngectomy & Bilateral Neck Dissection
439	Glorig	Hearing Aid Selection
447	Hantman	Newer Aids in Teaching Otology
464	Kasper	Dacryocystorhinostomy
469	Kinney	Hearing Impairments in Children
473	Lederer	Prevention of Otolaryngologic Misadventures
477	Lundy	Analgesia & Anesthesiology
484	Moore, P. M.	Cysts & Fistulas of Face & Neck
487	Neivert	Surgery of Bony Nasal Vault
488	Ogura	Laryngectomy & Neck Dissection
495	Rusk	Head-Low Position in Tonsillectomy
502	Simmons	Problem of Bleeding Patient
503	Simonton	Nasal Polyps
510	Suehs	Secretory Otitis Media
517	Van Alyea	Management of Sinusitis
521	Wanamaker	Pain on Side of Face

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
407	Anson & Bast	Anatomy of Ear & Temporal Bone
410	Baker	Chronic Nonspecific Laryngitis
412	Becker	Plastic Surgery of Protruding Ears
418	Canfield	Speech Audiometry
431	Erich	Fractures of Facial Bones
432	Farrior	Laryngectomy, Neck Resection & Cancer of Larynx
441	Goldman	Vasomotor Rhinitis
444	Gorney	Esophageal Strictures
445	Hamilton	Allergic Reactions to Drugs & Antibiotics.
460	Hunnicutt	The Chronically Congested Nose
461	Jones, E.	Allergic Rhinitis
463	Juers	Preservation of Hearing in Surgery
471	Kos	Stapedial Mobilization
474	Lempert	Temporal Bone Surgery
478	McHugh	Peripheral & Central Hearing Disorders in Children
479	McLaurin	Treatment of External Otitis
497	Schuknecht	Perceptive Deafness
514	Tolan	Treatment of Chronic Sinusitis
518	Von Leden & Moore, P.	Organic Voice Problems
519	Wallner	Effects of Tobacco on Respiratory Tract
524	Wynn	External Nasal Growths

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
404	Alfaro	Office Design & Equipment
409	Ashley	Respiratory Allergy
429	DeWeese	Problem of the Dizzy Patient
430	Dolowitz	Adrenal Steroids as Anti-Inflammatory Agents
438	Gill	Mycotic Infections
440	Glorig	The Noise Problem
443	Goodhill	Management of Tinnitus
454	Holinger & Johnston	Pediatric Laryngology
455	Hoople	Pitfalls in Office Audiometry
457	House	Stapes Mobilization Procedure
460	Hunnicutt	The Chronically Congested Nose
470	Kobrak	Treatment of Middle Ear Deafness
480	McNally	Labyrinthine Tests
481	Miller, A.	Laryngeal & Tracheal Emergencies in Infants & Children
488	Ogura	Laryngectomy & Neck Dissection
489	Pearlman	ENT Complaints in Old Age
494	Rinkel	Food Allergy
500	Senturia	Middle Ear Effusions
501	Shambaugh	Practical Office Routine
504	Sisson	Carcinoma of Maxillary Sinus
509	Sooy	Benign & Malignant Tumors of Nasal Cavities
511	Syracuse	Prevention of Postoperative Drooping of Nasal Tip
516	Tschiassny	Labyrinthine Function

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
417	Brodnitz	The Actor & Singer in ENT Practice
421	Costen	Mandibular Joint Syndrome
450	Higbee	Office Practice
464	Kasper	Dacryocystorhinostomy
469	Kinney	Hearing Impairments in Children
471	Kos	Stapedial Mobilization
473	Lederer	Prevention of Otolaryngologic Misadventures
484	Moore, P.M.	Cysts & Fistulas of Face & Neck
490	Perlman	Fluid in Middle Ear
491	Putney	Combined One-Stage Pharyngeal Diverticulectomy
496	Ryan	Headache
515	Tremble	Practical Therapeutics in Office Practice
522	Williams	Treatment of Meniere's Disease
523	Woodman	Glottic Obstruction

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
415	Bellucci	Surgery of Stapes & Oval Window
419	Cody	Eustachian Tube
422	Craddock	Hay Fever
426	Davison	Ethmoid Sinus Disease
431	Erich	Fractures of Facial Bones
433	Farrior	Fenestration Operation
442	Goldman	Diseases of Maxillary Sinus
444	Gorney	Esophageal Strictures
446	Hampsey	Sinusitis in Children
451	Hilger	The Injured Malar Bone
478	McHugh	Peripheral & Central Hearing Disorders in Children
483	Missal	Skin Testing
485	Morrison	The Pain in the Neck
495	Rusk	Head-Low Position in Tonsillectomy
497	Schuknecht	Perceptive Deafness
498	Sellers	Frontal Sinusitis: Acute & Chronic Infections
514	Tolan	Treatment of Chronic Sinusitis

News - - - Notes

PAN AMERICAN ASSOCIATION OF OPHTHALMOLOGY

Dr. Brittain F. Payne, New York, was elected president of the Pan American Association of Ophthalmology at the Fifth Pan American Congress of Ophthalmology in Santiago, Chile, January 8-14, 1956.

Dr. J. Wesley McKinney, Memphis, Tennessee, was elected executive secretary for countries north of Panama, succeeding Dr. Thomas D. Allen and Dr. Daniel Snydacker, both of Chicago. Dr. Snydacker has recently served as acting executive secretary for Dr. Allen. Dr. Jorge Balza, Buenos Aires, executive secretary for countries south of Panama, was re-elected.

Dr. Moacyr E. Alvaro, Sao Paulo, Brazil, president of the Association for the past four years and previously executive secretary for the countries to the south, was elected to the newly created office of executive director.

The Santiago Congress was attended by some 400 ophthalmologists, of whom about 150 were from the United States. There were three special guests from Europe: Dr. Edmond Velter, Paris; Dr. Frank S. Lavery, Dublin; and Count Hermenegildo Arruga, Barcelona.

The next meeting of the Congress will be the Fourth Interim Congress, in conjunction with the annual meeting of the National Society for the Prevention of Blindness at Hotel Statler in New York, April 7-10, 1957. The next regular Congress will be in Caracas, Venezuela, in 1960.

For the Interim Congress, three official subjects, to be discussed by invited speakers, have been selected. These are "Diseases of the Ocular Fundus," "Ophthalmic Surgery," and "Therapeutics in Present-Day Ophthalmology." "Free" papers on other topics may be included in the program as time allows. Dr. John M. McLean, professor of clinical surgery in ophthalmology, Cornell Medical College, New York, is chairman of the program committee. Those who wish to present papers are invited to communicate with Dr. McLean at The New York Hospital, 525 East 68th Street, New York 21.

A cinema program relating to the three official subjects is being planned under the chairmanship of Dr. Wendell L. Hughes, Professional Building, Hempstead, N. Y. Ophthalmologists who have suitable films should write to Dr. Hughes. There will also be a number of interesting exhibits.

Dr. Conrad Berens, former president of the Association, and Dr. Algernon B. Reese, chairman of the Fellowship Committee, both of New York, are serving as consultants on plans for the Congress.

Dr. Moacyr E. Alvaro, Sao Paulo, Brazil, past president and now executive director of the Association, is active in the preparations among Latin American ophthalmologists. He will head a large delegation of South American eye specialists from various southern countries.

Ophthalmologists planning to attend the Interim Congress should make their reservations with the Hotel Statler direct.

The Pan American Association of Ophthalmology will hold the annual cocktail party at 6:00 p.m., Sunday, October 14, 1956, at the Illinois Athletic Club, Chicago.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

At the Sixth International Congress of Otolaryngology, which will take place in Washington, D. C., May 5-10, 1957, three plenary sessions are scheduled for Monday, Wednesday and Friday mornings, May 6, 8 and 10. The subjects which have been selected and the internationally recognized authorities who will open the discussion are as follows:

Chronic Suppuration of the Temporal Bone

Anatomy and Etiology:
Marcus Diamant, Sweden

Genesis of Cholesteatoma:
Luzius Ruedi, Switzerland

Tympanoplasty:
Horst Wullstein, Germany

Collagen Disorders of the Respiratory Tract

Introduction:
Hans Selye, Canada

Upper Respiratory Tract:
Michele Arslan, Italy

Lower Respiratory Tract:
Leslie Gay, U.S.A.

Papilloma of the Larynx
Etiology:
Jo Ono, Japan
Diagnosis:
Plinio de Mattos Barretto, Brazil
Therapy:
F. C. W. Capps, England

ALUMNI LUNCHEONS

The Harry S. Gradle Alumni, comprising former residents of Michael Reese Hospital, will hold their annual Alumni Luncheon on Tuesday, October 9, 1956, at the Standard Club, Jackson Blvd. at Federal Court, Chicago.

The luncheon date is arranged so that members may attend their university or infirmary alumni dinners. Dr. Jack P. Cowen, 111 N. Wabash Ave., Chicago 2, is president of the Harry S. Gradle Alumni.

The Arlington C. Krause Luncheon will be held at the Captain's Cabin, Illinois Athletic Club, at 12:00 noon, Wednesday, October 17, 1956. The cost of the luncheon is $4.00 and does not include cocktails. Wives of guests will not be admitted. The secretary of the group is Paul G. Wolff, M.D., 1819 Broadway, Cape Girardeau, Mo.

THE AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct an examination October 8-11, 1956, at the Palmer House in Chicago.

Dr. Dean M. Lierle, University Hospitals, Iowa City, Iowa, is the secretary of the Board.

THE AMERICAN COLLEGE OF SURGEONS

The American College of Surgeons at the Annual Meeting in Chicago Nov. 3, 1955 elected Dr. Francis Adler of Philadelphia as a member of the Board of Governors to represent the Academy for the three-year period ending with the Annual Meeting, Oct. 5-10, 1958.

The others representing the American Academy of Ophthalmology and Otolaryngology are Dr. Derrick Vail (term expiring in 1956), and Dr. Shirley Harold Baron (term expiring in 1957).

The Council of the Academy at the Annual Meeting, Oct. 9-14, 1955, nominated for the term 1956-59 Dr. James McLaurin, Dr. Harry Schenck and Dr. Carl McCaskey.

FELLOWSHIPS IN OPHTHALMOLOGY

Twelve ophthalmologists have been appointed to serve on committees to select recipients for the residency fellowships in ophthalmology, recently established by the Guild of Prescription Opticians (TRANSACTIONS, Jan.-Feb., 1956). They are Alson E. Braley, M.D., Iowa City, Iowa; F. Phinizy Calhoun, Jr., M.D., Atlanta; David G. Cogan, M.D., Boston; Alfred J. Elliot, M.D., Toronto, Ont., Canada; R. M. Fasanella, M.D., New Haven, Conn.; R. E. Hannon, M.D., Cleveland; Michael J. Hogan, M.D., San Francisco; Frank W. Newell, M.D., Chicago; Shaler Richardson, M.D., Jacksonville, Fla.; Benjamin M. Rones, M.D., Washington, D. C.; Theodore E. Sanders, M.D., St. Louis; and Harold Schele, M.D., Philadelphia.

MASSACHUSETTS

Carroll R. Mullen, M.D., has been appointed professor and head of the Department of Ophthalmology at The Jefferson Medical College of Philadelphia.

NEW YORK

The Women's Medical Association of New York offers the Mary Putnam Jacobi Fellowship to a graduate woman physician, either American or foreign. This fellowship will start October 1, 1957 and will amount to $2000, $1000 being available October 1, 1957. The recipient of the fellowship will be expected to make a report to the committee at the end of the fourth month following which the second $1000 will be awarded subject to the approval of the committee. The fellowship is given for medical research, clinical investigation or postgraduate study in a special field of medicine.

Applications for this fellowship must be filed with the secretary of the committee by October 1, 1956 and will be acted upon by January 1, 1957. Application blanks may be obtained from the secretary of the committee and must be accompanied by (1) a statement from a

physician of a recent physical examination, (2) transcripts of her college and medical school records, (3) personal letters of recommendation from two or more physicians under whom she has studied, (4) a statement by the applicant of the problems she proposes to investigate or the study she plans to undertake, (5) a statement from the person under whom she proposes to study of his or her interest in her subject, (6) a recent photograph of the applicant together with a notation of her age. All of the above data must be at hand before the application will be considered.

The secretary is Ada Chree Reid, M.D., 118 Riverside Drive, New York 24.

OHIO

Ohio State University, Department of Ophthalmology, announces a course in orthoptic training to begin the first week of September 1956 and to continue for nine months. There will be no tuition fee, but the students must maintain themselves. Enrollment is limited to four.

PENNSYLVANIA

Albert P. Seltzer, M.D., has been appointed chief of the Department of Otolaryngology at Philadelphia General Hospital, Blockley Division.

POSITIONS AVAILABLE

An excellent opportunity is available in Aurora, Illinois, for a qualified E. E. N. T. man, due to the retirement of Dr. George A. Darmer who has maintained a practice there for forty years. Aurora, a city of 50,000 inhabitants, has only four ophthalmologists and otolaryngologists at the present time. Dr. Darmer will be happy to give full information to interested persons. His address is 401 Terminal Bldg., Aurora.

The Wichita Falls Clinic-Hospital has an opening for a certified or Board-eligible ophthalmologist; association with twenty-two specialists serving a college city of 110,000, with referred work from surrounding communities. Salary for first year with opportunity to enter partnership. Inquiries should be addressed to O. P. Moffitt, M.D., Wichita Falls Clinic-Hospital, Thirteen Hundred Eighth, Wichita Falls, Texas.

A certified or Board-eligible ophthalmologist or otolaryngologist is desired as an associate for Board-certified ophthalmologist or otolaryngologist in a well-established group. A physician who can work in either specialty is preferred. Interested persons should contact S. R. Bloomenthal, M.D., Sharon Medical Clinic, Ltd., 912 E. State St., Sharon, Pa.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date. Send to TRANSACTIONS, *American Academy of Ophthalmology and Otolaryngology,* 100 *First Avenue Bldg., Rochester, Minn.*

EXAMINING AND CERTIFYING BODIES

American Board of Ophthalmology

Chairman: Dr. Brittain F. Payne
Secretary: Dr. Merrill J. King, Box 236, Cape Cottage Branch, Portland 9, Maine
Practical Examination: October 20-24, 1956, St. Louis
Written Examination: January 21, 1957

American Board of Otolaryngology

President: Dr. LeRoy Schall
Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City, Iowa
Oral Examination: October 8-11, 1956; Palmer House, Chicago

American Board of Plastic Surgery

Chairman: Dr. James T. Mills
Secretary-Treasurer: Dr. Bradford Cannon, 330 Dartmouth St., Boston 16
Corresponding Secretary: Estelle E. Hillerich, 4647 Pershing Ave., St. Louis 8

Brazilian Board of Ophthalmology

Chairmen: Prof. Moacyr E. Alvaro
Prof. Cyro de Rezende
General Secretaries: Dr. Rubens Belfort Mattos, Caixa Postal 4086, Sao Paulo, Brazil
Dr. Sylvio de Almeida Toledo, Braulio Gomes 25, Sao Paulo, Brazil

The Royal College of Physicians and Surgeons of Canada

President: Dr. John Hepburn
Honorary Secretary: Dr. James H. Graham, 150 Metcalfe St., Ottawa 4, Ont., Canada

INTERNATIONAL

Concilium Ophthalmologicum Universale (International Council of Ophthalmology)

President: Sir Stewart Duke-Elder
Secretary: Dr. Edward Hartmann, 2 Ave. Ingres, Paris (16e), France
Time and Place: The Council meets at the time and place of each International Congress (usually every 4 years) and other years alternately in France and Great Britain.

Eighteenth International Congress of Ophthalmology

Secretary: Dr. Leon Coppez, 17 Ave. de Phorizon, Brussels, Belgium
Time and Place: September 1958; Brussels, Belgium

International Association of Secretaries of Ophthalmological and Otolaryngological Societies

President: Dr. Willard F. Goff
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Time and Place: October 16, 1956, Palmer House, Chicago

International Association for the Prevention of Blindness

President: Prof. A. Franceschetti
Secretary: Dr. J. B. Bailliart, 47 Rue de Bellechasse, Paris VII, France

International Broncho-Esophagological Society

President: Dr. Julio Cesar Barani
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia
Time and Place: August 31-September 2, 1956; Vienna. May 12-13, 1957; Philadelphia

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. Francis L. Lederer
Secretary: Dr. Louis Savitt, 3401 Lawrence Ave., Chicago 25

International Federation of Ophthalmological Societies

President: Sir Stewart Duke-Elder
Secretary: Prof. Dr. Edward Hartmann, 2 Ave. Ingres, Paris (16e), France
Time and Place: September 1958; Brussels, Belgium

International Organization Against Trachoma

President: Prof. G. B. Bietti
Secretary-General: D. T. Sedan, 94 rue Sylvabelle, Marseilles, France

Pan-American Association of Ophthalmology

President: Dr. Brittain F. Payne
Secretaries: For North of Panama: Dr. J. Wesley McKinney, Exchange Bldg., Memphis, Tenn.
For South of Panama: Dr. Jorge Balza, Belgrano 1675, Buenos Aires, Argentina
Time and Place: *Fourth Interim Congress;* April 7-10, 1957; Hotel Statler, New York
Sixth Pan-American Congress of Ophthalmology; 1960, Caracas, Venezuela. (Jointly with National Society for the Prevention of Blindness)

PAN-AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

President: Dr. Juan M. Tato
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz
Secretary: Dr. Paul H. Holinger, 700 North Michigan Ave., Chicago
Time and Place: May 5-10, 1957; Washington, D. C.

FOREIGN

Argentina

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

President: Dr. Jorge Balza
Secretary: Dr. Edgardo Manzitti, Santa Fé 1171, Buenos Aires
Time and Place: Third Wednesday of every month; Buenos Aires

Australia

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. Peter B. English
Secretary: Dr. Peter A. Rogers, 27 Commonwealth St., Sydney
Time and Place: July 23, 1956; Brisbane. October 1956; Melbourne

Austria

OSTERREICHISCHE OTO-LARYNGOLOGISCHE GESELLSCHAFT
(AUSTRIAN OTOLARYNGOLOGICAL SOCIETY)

President: Prof. Dr. Emil Schlander
Secretary: Dr. Julius Popper, Schottenring 17, Vienna I.
Time and Place: First Tuesday of the month; Vienna General Hospital, Alserstrasse 4, Vienna IX.

OSTERREICHISCHEN OPHTHALMOLOGISCHEN GESELLSCHAFT

President: Prof. Dr. Karl Lindner
Secretary: Dr. Franz Schwab, 1, Universitäts Augënklinik, Spitalgasse, 2, Vienna 9

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN

President: Prof. Dr. Josef Böck
Secretaries: Dr. Franz Schwab, and Dr. Rudolf Gittler, Alserstrabe 4
Time and Place: Second Monday of each month; 2, Universitäts-Augenklinik, Alserstrabe 4

Belgium

SOCIETE BELGE D'OPHTHALMOLOGIE

Secretary-General: Dr. M. Appelmans, 179 ave. Reulens, Louvain
Time and Place: November 25, 1956, Blvd. de Waterloo, 54, Brussels

Brazil

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA

President: Dr. Orlando Rebello
Secretaries: Dr. Renato Periera Machado, Dr. Carlos Henrique Bessa, Dr. Aderbal de Albuquerque Alves

CENTRO DE ESTUDOS DE OFTALMOLOGIA DE SAO PAULO

President: Dr. Francisco Amendola
Secretary: Dr. Rubens Belfort Mattos, Caixa Postal 4086, Sao Paulo
Time and Place: Every Tuesday evening; Rua Condessa, Sao Joaquim 288, Sao Paulo

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO

President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevedo, Xavier de Toledo 71, 1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OFTALMOLOGIC E OTORRINOLARINGOLOGIA
DO RIO GRANDE DO SUL

President: Dr. Paulo Fernando Esteves
Secretary: Dr. Carlos Buede, Rua Uraguay N. 240, Ed. Piratini 10.° andar, Porto Alegre, Rio Grande do Sul

Canada

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

Chairman: Dr. John Clement McCulloch, 830 Medical Arts Bldg., Toronto, Ont.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Henri Pichette
Secretary: Dr. R. G. C. Kelly, 90 St. Clair Ave. West, Toronto 7, Ont.

SOCIETE CANADIENNE D'OTOLARYNGOLOGIE
(CANADIAN OTOLARYNGOLOGICAL SOCIETY)

President: Dr. D. S. Gorrell
Secretary: Dr. G. Arnold Henry, 328 Medical Arts Bldg., Toronto, Ont.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. A. L. Guertin
Secretary: Dr. William Turnbull, 1414 Drummond St., Montreal
Time and Place: Second Thursday in October; Royal Victoria Hospital. Second Thursday in December; Notre Dame Hospital

NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. G. Holland
Secretary: Dr. C. F. Keays, 324 Spring Garden Rd., Halifax
Time and Place: November 1956; Lord Nelson Hotel, Halifax

OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY OF ALBERTA

President: Dr. E. F. Foy
Secretary: Dr. M. R. Marshall, North Pavilion, University Hospital, Edmonton, Alta.
Time and Place: Spring, 1957; Edmonton, Alta.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. G. A. Thompson
Secretary: Dr. J. L. Burns, 1421 Danforth Ave., Toronto, Ont.
Time and Place: First Monday, October to April; Osler Hall, Toronto Academy of Medicine

Chile

SOCIEDAD CHILENA DE OFTALMOLOGIA

President: Dr. Alberto Gormaz B.
Secretary: Dr. Carlos Charlin V., Mc. Iver No. 175, Santiago
Place: Auditorium, Clinica Oftalmalogica Hospital del Salvador, Santiago

Colombia

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARYNGOLOGIA

President: Dr. Francisco Vernaza
Secretary: Dr. Félix E. Lozano Diaz, Apartado Aèreo 5601, Bogota

Cuba

SOCIEDAD CUBANA DE OFTALMOLOGIA

President: Dr. Rafael Labrador Fuiz
Secretary: Dr. Guillermo De La Torre, Calle G. No. 506, Vedado, Havana
Time and Place: First Thursday bi-monthly; Malecon 61, Bajos, Havana

Denmark

OPHTHALMOLOGICAL SOCIETY IN COPENHAGEN, DENMARK

President: Dr. Børge Lawatz

Egypt

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Dr. Fattouh Mohammad Fattouh
Honorary Secretary: Dr. Sabri Kamel, Dar El Hekmah, 42 Kasr-El-Ainy St., Cairo
Time and Place: March 1957, Memorial Institution for Ophthalmic Research, Giza

France

SOCIETE FRANCAISE D'OPHTALMOLOGIE

Secretary-General: Dr. Marcel Kalt, 81 rue Saint Lazare, Paris (9e)

Germany

GERMAN OPHTHALMOLOGICAL SOCIETY
UNITED CONGRESS

Time and Place: September 3-6, 1956; Heidelberg, Aula of the new University

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG

President: Prof. Dr. K. Lindner
Secretary: Prof. Dr. E. Engelking, Universitäts-Augenklinik, Heidelberg
Time and Place: September 2-6, 1956; Heidelberg

Great Britain

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS

President: Mr. I. Simson Hall, F.R.C.S.E.
Secretary: Mr. Myles L. Formby, F.R.C.S., 45 Lincoln's Inn Fields, London W.C.2.

FACULTY OF OPHTHALMOLOGISTS

President: Dr. John Marshall
Secretary: Mr. E. F. King, F.R.C.S., 45 Lincoln's Inn Fields, London W.C. 2.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: J. J. Healy, M.B., Ch.B.
Secretaries: C. A. Cook, M.C., G.M., F.R.C.S.; J. R. Hudson, F.R.C.S., 45 Lincoln's Inn Fields, London, W.C. 2

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: R. C. Davenport, F.R.C.S.
Honorary Secretaries: J. E. M. Ayoub, F.R.C.S., and E. S. Perkins, F.R.C.S., 1 Wimpole St., London, W.1.
Time and Place: Second Thursday of month from October to June; 1 Wimpole St, London, W.1.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Mr. P. Jameson Evans
Secretaries: Mr. J. J. Roper-Hall and Mr. W. Martin Walker, Birmingham and Midland Eye Hospital, Church St., Birmingham 3
Time and Place: October-June, Tuesday, Thursday, Friday or Saturday according to programme director; Birmingham Midland Eye Hospital, Church St., Birmingham 3

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: J. W. Tudor-Thomas, F.R.C.S.
Honorary Secretary and Treasurer: Ian C. Fraser, F.R.C.S., 21 Dogpole, Shrewsbury, Shropshire
Time and Place: July 2-4, 1956; School of Physiology, Oxford

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. John Marshall
Secretary: Dr. John D. Fraser, 31 Kingsborough Gardens, Glasgow
Time and Place: October 27, 1956; Glasgow

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: W. H. Summerskill, M.B., B.S., D.O. (Oxon.)
Secretary: Nigel Cridland, D.M., D.O., (Oxon.)., 25 Craneswater Park, Southsea, Hants.
Time and Place: September 29; Royal Berkshire Hospital, Reading. October 20; Portsmouth and Southern Counties, Eye and Ear Hospital, Portsmouth

India

ALL-INDIA OPHTHALMOLOGICAL SOCIETY

President: Capt. K. Sen
Secretaries: Dr. S. N. Cooper and Dr. Y. K. C. Pandit, Laud Mansion 21, Queen's Rd., Bombay 4.

BOMBAY OPHTHALMOLOGISTS' ASSOCIATION

President: Dr. D. S. Sardesai
Secretaries: Dr. B. D. Telang and Dr. Y. K. C. Pandit, 395 Lamington Rd., Bombay 4.
Time and Place: 7:30 p.m., third Wednesday of each month; the seven ophthalmic hospitals of Bombay by rotation

OPHTHALMOLOGICAL SOCIETY OF BENGAL

President: Capt. K. Sen
Secretaries: Dr. S. Bagchi and Dr. M. Sengupta, 94 Chittaranjan Ave., Calcutta

Ireland

IRISH OPHTHALMOLOGICAL SOCIETY

President: Dr. Frank Lavery
Secretary: Dr. L. B. Somerville-Large, 2 Fitzwilliam Place, Dublin

Mexico

SOCIEDAD MEXICANA DE OFTALMOLOGIA

President: Dr. Manuel de Rivas Cherif
Secretary: Dr. Emilio Remolina Lopaz, Apartado Postal No. 8112, Mexico, D.F.
Time and Place: 8:30 p.m., first Tuesday of each month; Gral. Prim 47

The Netherlands

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP

President: Prof. Dr. A. Hagedoorn
Secretary: Dr. P. G. Binkhorst, Sweelincklaan 20, Bilthoven

New Zealand

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

President: Dr. J. Monro
Secretary: Dr. C. C. Ring, 1A Alfred St., Auckland
Time and Place: July 1956; Wellington

Norway

NORSKE OYELEGERS FORENING
(SOCIETY OF NORWEGIAN OPHTHALMOLOGISTS)

President: Dr. Ludvig Wirsching
Secretary: Dr. med. H. P. Petersen, Rikshospitalet (Oyenavdelingen) Oslo

Paraguay

SOCIEDAD DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA DEL PARAGUAY

President: Dr. Crispin Insaurralde
Secretary: Dr. Honorio Campuzano, Coronel Bogado 492, Asuncion
Place: Asuncion

Philippines

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Carlos V. Yambao
Secretary-Treasurer: Dr. Jesus V. Tamesis, North General Hospital, Manila

Portugal

SOCIEDADE PORTUGUSEA DE OTORRINOLARINGOLOGIA E DE BRONCHO-ESOFAGOLOGIA

President: Dr. Alberto Luis de Mendonca
Secretaries: Dr. Antonio da Costa Quinta and Dr. Albano Coelho, Avenida da Liberdade, 65, Lisbon 1

Union of South Africa

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

President: Dr. J. K. de Kock
Secretary: Dr. J. Guilliaume Louw, 17 Church Square, Cape Town

Spain

SOCIEDAD OFTALMOLOGICA HISPANO-AMERICANA

President: Dr. Manuel Lopez Enriquez
Secretary: Dr. Carlos Costi Garcia de Tunon, Montalban, 7, Madrid

Sweden

SVENSKA OGONLAKAREFORENINGEN, SVENSKA LAKARESALLSKAPETS SEKTION FOR OFTALMOLOGI

President: Prof. G. Karpe
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm So.

NATIONAL

American Academy of Ophthalmology and Otolaryngology

President: Dr. A. C. Furstenberg
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Bldg., Rochester, Minn.
Time and Place: October 14-19, 1956; Palmer House, Chicago

American Broncho-Esophagological Association

President: Dr. Daniel S. Cunning
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3

American Laryngological Association

President: Dr. Bernard J. McMahon
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3

American Laryngolocical, Rhinological and Otological Society, Inc.

President: Dr. Percy E. Ireland
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

Eastern Section

Chairman: Dr. John A. Murtagh
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3

Middle Section

Vice-President: Dr. William H. Craddock
Secretary: Dr. Charles E. Kinney, 10515 Carnegie Ave., Cleveland 6

Southern Section

Vice-President: Dr. J. M. Robison
Secretary: Dr. James A. Harrill, Bowman Gray School of Medicine, Winston-Salem, N. C.

Western Section

Vice-President: Dr. David D. DeWeese
Secretary: Dr. Francis Sooy, 490 Post St., San Francisco

American Medical Association, Section on Ophthalmology

Chairman: Dr. Algernon B. Reese
Secretary: Dr. Harold G. Scheie, 313 South 17th St., Philadelphia 3
Time and Place: Clinical meeting, November 27-31, 1956; Seattle. Annual meeting, June 3-7, 1957; New York

American Medical Association, Section on Otolaryngology

Chairman: Dr. John R. Lindsay
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Time and Place: Annual meeting, June 3-7, 1957; New York

American Ophthalmological Society

President: Dr. Alan C. Woods
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th St., New York 19

American Otological Society, Inc.

President: Dr. William J. McNally
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14

American Otorhinologic Society for Plastic Surgery

President: Dr. Joseph Gilbert
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16
Place: New York

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Dean M. Lierle
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2
Time and Place: October 19, 1956; Palmer House, Chicago

Association for Research in Ophthalmology, Inc.

Chairman: Dr. William B. Clark
Secretary: Dr. Lorand V. Johnson, 10515 Carnegie Ave., Cleveland 6
Time and Place: June 3-7, 1957, New York

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow
Executive Director: Dr. Franklin M. Foote, 1790 Broadway, New York 19
Time and Place: April 7-10, 1957 (Jointly with Fourth Interim Congress of the Pan-American Association of Ophthalmology); New York

Puerto Rico Medical Association, Section on Ophthalmology and Otolaryngology

President: Dr. Roberto Buxeda
Secretary: Dr. Guillermo Pico, 654 Ponce de Leon Ave., Santurce
Time and Place: Every other month; Puerto Rico Medical Association Bldg., Santurce

Puerto Rico Ophthalmological Society

President: Dr. Roberto Buxeda
Secretary-Treasurer: Dr. Guillermo Pico, 654 Ponce de Leon Ave., Santurce
Time and Place: Every other month; Puerto Rico Medical Association Bldg, Santurce

Society of Military Ophthalmologists

President: Col. Victor Byrnes
Secretary-Treasurer: Capt. James A. Stokes, M.C., Walter Reed Army Hospital, Washington 12, D. C.
Time and Place: October 16, 1956; Palmer House, Chicago

REGIONAL

Ark-La-Tex Oto-Ophthalmic Society

President: Dr. J. L. Custer
Secretary: Dr. W. W. Hart, Medical Arts Bldg., Shreveport, La.
Time and Place: First Monday of month, except January, June, July, September; Confederate Memorial Hospital

Hawaii Eye, Ear, Nose and Throat Society

President: Dr. T. W. Cowan
Secretary: Dr. Robert H. Lee, 1443 Nuuanu Ave., Honolulu 17
Time and Place: 6:30 p.m. (dinner meetings), third Thursday of each month, October-June; Pacific Club, Honolulu

Inter-Mountain Oto-Ophthalmological Society

President: Dr. M. Mairhead
Secretary: Dr. Alan S. Crandall, 141 E. Second St. South, Salt Lake City 1
Time and Place: Third Monday, September-May; Salt Lake City

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. H. K. Rouse
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

New England Ophthalmological Society

President: Dr. Merrill J. King
Secretary: Dr. Karl Riemer, 403 Commonwealth Ave., Boston
Time and Place: Third Wednesday, November through April; Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston

New England Oto-Laryngological Society

President: Dr. Burton E. Lovesey
Secretary: Dr. Burton E. Lovesey, 15 West Central St., Natick, Mass.
Time and Place: Three times a year, Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston

Pacific Coast Oto-Ophthalmological Society

President: Dr. H. Leroy Goss
Secretary: Dr. Homer E. Smith, 508 E. South Temple, Salt Lake City, Utah
Time and Place: April 7-11, 1957, Coronado, California

Sioux Valley Academy of Ophthalmology and Otolaryngology

President: Dr. F. A. Finnegan
Secretary: Dr. Sidney F. Becker, B-1600 S. Western Ave., Sioux Falls, S. D.
Time and Place: February 1957; Sioux City, Iowa

Southern Medical Association, Section on Ophthalmology and Otolaryngology

Chairman: Dr. William J. G. Davis
Secretary: Dr. G. S. Fitz-Hugh, 104 Market St., Charlottesville, Va.
Time and Place: November 12-15, 1956, Washington, D. C.

Wisconsin-Upper Michigan Society of Ophthalmology and Otolaryngology

President: Dr. William W. Ford
Secretary: Dr. G. L. McCormick, 650 S. Central Ave., Marshfield, Wis.

STATE AND LOCAL

Alabama

Alabama Academy of Ophthalmology and Otolaryngology

President: Dr. John K. Lingo
Secretary: Dr. Eugene Callaway, Jr., Selma
Time and Place: January 17, 1957, Point Clear

Birmingham Eye, Ear, Nose and Throat Society

President: Each member, in alphabetical order
Secretary: Dr. David A. McCoy, 501 Woodward Bldg., Birmingham
Time and Place: 6:30 p.m., second Tuesday of each month, September through April; The Club, Top of Red Mountain, Birmingham

Arkansas

Arkansas Medical Society, Eye, Ear, Nose and Throat Section

President: Dr. W. F. Schwarz
Secretary: Dr. James L. Smith, 623 Woodlane, Little Rock

California

California Medical Association, Eye, Ear, Nose and Throat Section

Chairman: Dr. Robert N. Shaffer
Secretary: Dr. Channing Hale, 174 Nehama, Pomona

Alta California Ophthalmologic Society

President: Dr. John A. Berg
Secretary: Dr. Robert L. Alexander, 1127 Eleventh St., Sacramento
Time and Place: Third Thursday of every month except July and August; Sutter Club, Sacramento

East Bay Ophthalmological Society

President: Dr. Dudley Bell
Secretary: Dr. Arthur F. Steinmetz, 1237 B St., Hayward
Time and Place: Every three months, different locations

LOS ANGELES OPHTHALMOLOGICAL JOURNAL CLUB

President: Dr. Jacob M. Kline
Secretary: Dr. William Feldman, 7705 Leville Ave., Los Angeles
Place: Los Angeles County Medical Bldg.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Leland R. House
Secretary: Dr. Sol Rome, 1917 Wilshire Blvd., Los Angeles 5
Time and Place: 6:00 p.m., fourth Monday, September-June, Otolaryngology; 6:00 p.m., first Thursday, September-June, Ophthalmology; Los Angeles County Medical Assn. Bldg., 1925 Wilshire Blvd.

SAN DIEGO OPHTHALMOLOGICAL SOCIETY

President: Dr. H. R. Merrill
Secretary: Dr. A. B. Root, 3315 Fourth Ave., San Diego 3
Time and Place: 6:30 p.m., second Monday of each month; University Club

SAN FRANCISCO COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman and Secretary: Dr. Harold O. Parkinson, 760 Market St., San Francisco
Time and Place: 9:15 p.m., first Tuesday of each month, April, August and December, San Francisco Medical Society Bldg., 250 Masonic Ave.

SAN FRANCISCO OPHTHALMOLOGICAL ROUND TABLE

President: Dr. George S. Campion
Secretary: Dr. Albert J. Brinckerhoff, 490 Post St., San Francisco
Time: Three times a year

Colorado

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Thomas Van Bergen
Secretary: Dr. James C. Strong, Jr., 227 Sixteenth St., Denver
Time and Place: Third Saturday of each month, September to May; Colorado Medical Center, Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY

President: Dr. Donald F. Page
Secretary: Dr. Sidney H. Fieman, 730 Republic Bldg., Denver 2
Time and Place: 8:00 p.m., first Saturday of each month; Colorado General Hospital

Connecticut

CONNECTICUT STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Max Alpert
Secretary-Treasurer: Dr. Frederick Wies, 225 Bradley St., New Haven

Florida

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Charles W. Boyd
Secretary: Dr. Kenneth S. Whitmer, 550 Brickell Ave., Miami

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. Hollender
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami
Time and Place: February, May, October and December; Seven Seas Restaurant

Georgia

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. M. Barton
Secretary: Dr. Walter P. Rhyne, Box 206, Albany
Time and Place: April 1957; General Oglethorpe Hotel, Wilmington Island, Savannah. Cruise from Charleston, S. C., to Nassau, The Bahamas.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William T. Edwards
Secretary-Treasurer: Dr. Jack Stokes, 512 Medical Arts Bldg., 384 Peachtree St., N.E., Atlanta
Time and Place: Fourth Monday of each month, Fulton County Academy of Medicine, Atlanta

Illinois

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Owen
Secretary: Dr. Clarence A. Fleischli, 520 East Allen St., Springfield
Time and Place: September 1956; Peoria

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond W. Kerwin
Secretary: Dr. Stanton A. Friedberg, 122 S. Michigan Ave., Chicago 3
Time and Place: First Monday of each month at various places

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Kenneth L. Roper
Secretary-Treasurer: Dr. Joseph S. Haas, 109 N. Wabash Ave., Chicago 2
Time and Place: 7:30 p.m., third Monday of each month, October to May; Chicago-Illini Union, 715 S. Wood St.

Indiana

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. P. Clark
Secretary: Dr. John J. Flick, 1443 N. Pennsylvania St., Indianapolis
Time: Annually in May

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. David E. Jones
Secretary: Dr. Lewis E. Morrison, 603 Hume Mansur Bldg., Indianapolis 4
Time and Place: 6:30 p.m., second Thursday of each month during Winter; Athletic Club

Iowa

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Carl Noe
Secretary: Dr. Arthur C. Wise, 212 Savings and Loan Bldg., Iowa City

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert P. Updegraff
Secretary: Dr. C. C. Walker, 304 Bankers Trust Bldg., Des Moines, Iowa
Place: Des Moines Club

Kansas

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David P. Trimble
Secretary: Dr. Max Lake, Salina

Kentucky

LOUISVILLE EYE AND EAR SOCIETY

President: Dr. Charles Bloch
Secretary: Dr. George I. Uhde, 610-613 Brown Bldg., Louisville 2
Time and Place: Third Thursday of alternate months; at various places

Maryland

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION

Chairman: Dr. Herman K. Goldberg
Secretary: Dr. Charles Iliff, 14 W. Mt. Vernon Place, Baltimore
Time and Place: Third Thursday of November, January and March; Medical and Surgical Faculty Bldg., 1211 Cathedral St., Baltimore

BALTIMORE EAR, NOSE AND THROAT SOCIETY

President: Dr. Theodore A. Schwartz
Secretary: Dr. Alvin P. Wenger, 1100 N. Charles St., Baltimore
Time and Place: Second Tuesday in November, January and March; Johns Hopkins Club

Massachusetts

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Edgar M. Holmes
Secretary: Dr. John E. McKeigue, 94 Pleasant St., Arlington

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC SOCIETY

President: Dr. Albert E. Sloane
Secretary: Dr. Thomas P. White, 520 Commonwealth Ave., Boston

Michigan

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Bldg., Lansing (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Kenneth Lowe
Secretary: Dr. Wilfrid Haughey, 610 Post Bldg., Battle Creek
Time and Place: Four times a year in different cities in Michigan

DETROIT OPHTHALMOLOGICAL CLUB

Secretary: Dr. Frederick A. Lauppe, 1801 David Whitney Bldg., Detroit 26
Time: Each month, November through April

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Harold F. Falls
Secretary: Dr. James Olson, 1005 Mutual Bldg., 28 W. Adams, Detroit
Time and Place: 8:00 p.m., fourth Thursday of each month, The Harmonie Club, 267 E. Grand River

DETROIT OTOLARYNGOLOGICAL SOCIETY

President: Dr. James E. Croushore
Secretary: Dr. Lyle Waggoner, 404 David Whitney Bldg., Detroit 26
Time and Place: Third Wednesday of each month, Wayne County Medical Society

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Blackhurst
Secretary: Dr. Arno Weiss, 308 Eddy Bldg., Saginaw
Time and Place: Second Tuesday of each month, Bancroft Hotel, Saginaw

Minnesota

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Karl E. Sandt
Secretary: Dr. Malcolm A. McCannel, 325 Doctors Bldg., Minneapolis
Time and Place: Second Friday of each month; alternating in Minneapolis and St. Paul

Missouri

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Donald O. Howard
Secretary: Dr. Dick H. Underwood, 1109 Bryant Bldg., Kansas City
Time and Place: Third Thursday of the month, Kansas University Medical Center

EAR, NOSE AND THROAT CLUB OF ST. LOUIS

Chairman: Dr. L. W. Dean, Jr.
Secretary: Dr. Harry N. Glick, 1504 South Grand Blvd., St. Louis 4
Time and Place: Third Wednesday in November, January, March and May; University Club

ST. LOUIS OPHTHALMOLOGICAL SOCIETY

President: Dr. William H. Bryan
Secretary: Dr. Philip T. Shahan, 100 N. Euclid, St. Louis 8
Time and Place: 5:30 p.m., fourth Thursday in October, November, January, February, March and April; Park Plaza Hotel, 220 N. Kingshighway

Montana

MONTANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John J. Hammerel
Secretary: Dr. James D. Morrison, P. O. Box 1856, Billings
Time and Place: Annual meeting, September 13-15, 1956; Great Falls

Nebraska

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. E. Koebbe
Secretary-Treasurer: Dr. Paul L. Peterson, 702 Sharp Bldg., Lincoln

OMAHA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. George T. Alliband
Secretary-Treasurer: Dr. Stanley M. Truhlsen, 1500 Medical Arts Bldg., Omaha
Place: The Omaha Club

New Jersey

ACADEMY OF MEDICINE OF NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Edward Ehrlich
Secretary: Dr. William A. Sturman, 219 South Harrison St., East Orange
Time and Place: 8:45 p.m., four times a year; Academy Bldg., Newark Eye and Ear Hospital, Newark

MEDICAL SOCIETY OF NEW JERSEY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Joseph R. Weintrob
Secretary: Dr. John Scillieri, 660 Broadway, Patterson 4

NEW JERSEY OPHTHALMOLOGICAL SOCIETY

President: Dr. George P. Meyers
Secretary: Dr. Alfonse Cinotti, 3285 Hudson Blvd., Jersey City
Time and Place: November; Princeton Inn, Princeton. May; Atlantic City

NEW JERSEY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Edgar P. Cardwell
Secretary: Dr. Albert F. Moriconi, 438 Hamilton Ave., Trenton 9

HUDSON COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William McKeever
Secretary: Dr. Benjamin Jaffe, 566 Bergen Ave., Jersey City
Time and Place: 9:00 p.m., second Monday of each month; Pete and Dominicks Restaurant, Jersey City

New Mexico

NEW MEXICO OPHTHALMOLOGICAL SOCIETY

President: Dr. Howard B. Peck
Secretary: Dr. J. A. Dillahunt, 106 Girard Blvd. S.E., Albuquerque
Time: Semi-annually at the call of the President

New York

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

President: Dr. Maynard C. Wheeler
Secretary: Dr. Harold W. Brown, 15 Park Ave., New York
Time and Place: 8:15 p.m., third Monday of each month; New York Academy of Medicine, 2 E. 103rd St.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLARYNGOLOGY

Chairman: Dr. Otto C. Risch
Secretary: Dr. Ernest A. Weymuller, 69 East 76th St., New York
Time: 3rd Wednesday of each month

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Max Chamlin
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24
Time and Place: 8:00 p.m., first Monday of the month; New York Academy of Medicine, 2 E., 103rd St.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Lee Stoner
Secretary: Dr. George A. Sisson, 713 East Genesee St., Syracuse
Time and Place: February, May, August and November; The University Club, Syracuse

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Henry Little
Secretary-Treasurer: Dr. Frank C. Furlong, 713 Union St., Schenectady
Time and Place: 7:00 p.m., first Thursday of each month; alternating at Albany, Troy and Schenectady

ALUMNI ASSOCIATION OF THE NEW YORK EYE AND EAR INFIRMARY

President: Dr. Byron Smith
Secretary: Dr. Alfonse Cinotti, 3285 Hudson Blvd., Jersey City
Place: New York Eye and Ear Infirmary, 218-2nd Ave., New York 3

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Louis Freimark
Secretary-Treasurer: Dr. Nicholas P. Tantillo, 1 Hanson Pl., Brooklyn 17
Time and Place: Third Thursday in October, December, February and April; Kings County Medical Society, 1313 Bedford Ave., Brooklyn

BUFFALO OPHTHALMOLOGICAL CLUB

President: Dr. Elizabeth P. Olmsted
Secretary-Treasurer: Dr. Harold E. Wass, 840 West Delavan Ave., Buffalo 9

NASSAU OPHTHALMOLOGICAL SOCIETY

President: Dr. Conrad Berens
Secretary-Treasurer: Dr. Arthur E. Merz, 821 Franklin Ave., Garden City, L. I.
Time and Place: Monday evenings, February, April, September and November; at various places

North Carolina

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James C. Peele
Secretary: Dr. James D. Stratton, 1012 Kings Drive, Charlotte 7
Time and Place: September 16-19, 1956; Asheville

Ohio

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. F. A. Johnson
Secretary: Dr. John Scuderi, 143 Portage Trail, Cuyahoga Falls
Time and Place: 6:30 p.m., first Monday of every other month; Akron City Club

CINCINNATI OPHTHALMOLOGIC CLUB

Executive Committee: Dr. Donald J. Lyle, Dr. Charles K. W. Ascher, Dr. Ira A. Abrahamson, Sr.
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Bldg., Cincinnati 2
Time and Place: Second Wednesday night of month, October through May; Vernon Manor Hotel

CINCINNATI OTOLARYNGOLOGICAL SOCIETY

President: Dr. Erwin S. Ross
Secretary: Dr. Joseph A. Lane, 912 Provident Bank Bldg., Cincinnati
Time and Place: 6:30 p.m., third Monday of each month; University Club

CLEVELAND OPHTHALMOLOGICAL CLUB

President: Dr. Webb P. Chamberlain, Jr.
Secretary: Dr. Arthur D. Collins, 629 Euclid Ave., Cleveland 14
Time: Second Tuesday of November, January, February and April

CLEVELAND OTOLARYNGOLOGICAL CLUB

President: Dr. William F. Hulse
Secretary-Treasurer: Dr. Fred Alexander, 10515 Carnegie Ave., Cleveland 6
Time: Four times a year

COLUMBUS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. E. Arrington
Secretary: Dr. Trent W. Smith, 327 E. State St., Columbus 15
Time: First Monday of month, September through April

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Robert L. Willard
Secretary: Dr. Max F. Sudhoff, 316 Michigan Ave., Toledo 2
Time and Place: Third Tuesday of each month, October through May; Toledo Club

Oklahoma

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. A. Watson
Secretary: Dr. Richard Wyrick, 3832 N. W. 51 Pl., Oklahoma City 12
Time and Place: 6:30 p.m., first Tuesday of each month; University of Oklahoma Medical School

Oregon

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ralph N. Westfall
Secretary: Dr. Charles W. Browning, 919 Taylor St., Portland 5
Place: Henry Thiele Restaurant, 23 and West Burnside, Portland

Pennsylvania

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. William T. Hunt, Jr.
Secretary: Dr. Daniel S. De Stio, 1006 Highland Bldg., Pittsburgh 6

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. H. H. Robb
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Ave., DuBois
Time: Third Thursday of April and October

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

President: Dr. Isaac S. Tassman
Secretary: Dr. William E. Krewson, III, 1930 Chestnut St., Philadelphia 3
Time and Place: Third Thursday of every month, October-April; College of Physicians Bldg., 19 So. 22nd St., Philadelphia 3

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Wilfred E. Fry
Secretary: Dr. Thomas H. Cowan, 1930 Chestnut St., Philadelphia 3
Time and Place: First Thursday of each month, October through April; County Medical Society Bldg., 21st and Spruce Streets, Philadelphia

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. William J. Hitschler
Secretary: Dr. Joseph P. Atkins, 3400 Spruce St.
Time: First Tuesday of month, November through May

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Thomas G. McLellan
Secretary: Dr. Gertrude W. Baldwin, Greensburg
Time and Place: 8:00 p.m., third Monday of month, September through May; Mellon Institute

PITTSBURGH OTOLOGICAL SOCIETY

President: Dr. Louis L. Friedman
Secretary: Dr. John Dickinson, Mercy Hospital
Time: Quarterly

READING EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John J. Penta
Secretary: Dr. James H. Parker, Jr., 238 N. 5th St.
Time and Place: Third Wednesday of each month, Wyomissing Club, Reading

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

President: Dr. Vincent Gallizi
Secretary: Dr. Albert J. Abbott, 203 E. Green St., Nanticoke
Time and Place: Last Tuesday of each month; Luzerne County Medical Society Bldg.

South Carolina

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. N. O. Eaddy
Secretary: Dr. Roderick Macdonald, 330 East Main St., Rock Hill
Time and Place: September 17-19, 1956; Asheville, N. C. (Jointly with N.C.E.E.N.T. Society)

Tennessee

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Hurbert Duncan
Secretary: Dr. Thomas Bryan, 2105 Hayes St., Nashville
Time and Place: 1957; Nashville

CHATTANOOGA AND HAMILTON COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

Chairman and Secretary: Dr. I. L. Arnold, 702 Medical Arts Bldg., Chattanooga
Time and Place: 6:30 p.m., second Thursday in each month, Mountain City Club

KNOXVILLE SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Olin Ballou
Secretary: Dr. J. E. Campbell, Jr., 406 W. Church Ave.
Time and Place: 6:30 p.m., first Monday of each month, September through May; Hotel Farragut

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis 3
Time and Place: Second Tuesday of each month; Memphis Eye, Ear, Nose and Throat Hospital, 1060 Madison Ave.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Fred Harned
Secretary-Treasurer: Dr. Ralph Rice, Bennie Dillon Bldg.
Time and Place: Third Monday evening of month, October through May; James Robertson Hotel

Texas

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kell Cox
Secretary: Dr. Gatlin Mitchell, 1604 Medical Arts Bldg., Fort Worth
Time and Place: December 7-8, 1956; Dallas

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President: Dr. Edwin G. Grafton
Secretary-Treasurer: Dr. Hal W. Maxwell, 624 Medical Arts Bldg., Dallas 1
Time: First Tuesday of each month except July and August

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harold Beasley
Secretary: Dr. J. Robert Harris, 1016 W. Cannon
Time and Place: First Friday of each month, except July, August and September; St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION

President: Dr. Thomas Vanzant
Secretary: Dr. Fred R. Guilford, 509 Hermann Professional Bldg.
Time and Place: 6:30 p.m., second Thursday of each month; Doctors' Club

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Thomas P. O'Brien
Secretary: Dr. Joseph A. Abbey, 2451 Morgan, Corpus Christi
Time and Place: Privateer Club, Corpus Christi

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Chester L. Mohle
Secretary: Dr. Graham B. Milburn, 1104 Nix Professional Bldg.
Time: Third Tuesday of each month; San Antonio Country Club

Virginia

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ben Sheppard
Secretary: Dr. Maynard P. Smith, 600 Professional Bldg., Richmond

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ed Bryce
Secretary: Dr. Charles N. Romaine, IV, 1001 West Franklin St., Richmond 20
Time: First Tuesday of January, March, May and October

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. M. H. Hood
Secretary: Dr. John G. Sellers, 413 Medical Arts Bldg., Norfolk
Time and Place: October, February and May, Tidewater

Washington

WASHINGTON STATE MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

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Secretary-Treasurer: Dr. Claude K. Miller, 601 Douglas St., Wenatchee

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Russell T. Horsfield
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle 1
Time and Place: Third Tuesday of February, March, April, May, October and November; usually in Seattle

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert C. Maher
Secretary: Dr. Philip B. Green, Old National Bank Bldg.
Time and Place: 8:00 p.m., fourth Tuesday of each month; Blood Bank Bldg.

Washington, D. C.

SECTION ON OPHTHALMOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

President: Dr. William D. Foote
Secretary-Treasurer: Dr. M. Noel Stow, 1746 "K" St., N. W., Washington, D.C.
Time and Place: Every other month, October through April; Kennedy-Warren Apts.

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

President: Dr. Jack Levine
Secretary: Dr. James J. McFarland, 1150 Connecticut Ave. N. W.
Time and Place: Second Tuesday, every other month, October through May; Army-Navy Club

West Virginia

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ben W. Bird
Secretary-Treasurer: Dr. Frederick C. Reel, 404 Medical Arts Bldg., Charleston

Wisconsin

MADISON, WISCONSIN, EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Mark E. Nesbit
Secretary: Dr. Donald A. Peterson, 224 W. Washington Ave.
Time and Place: 6:15 p.m., first Tuesday of each month, October through May; Madison Club

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Donald T. Hughson
Secretary: Dr. E. Franklin Carl, 606 W. Wisconsin Ave., Milwaukee
Time and Place: 6:00 p.m., fourth Tuesday of each month; University Club of Milwaukee

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